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(REPRINTS)

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A HISTORY OF THE DEPARTMENT OF NEUROLOGY OF THE MEDICAL SCHOOL OF THE UNIVERSITY OF PENNSYLVANIA

By Charles K. Mills, M.D., Professor of Neurology.

Early Teaching of Neurology and Psychiatry in The University.

As an introduction to the history of the department of neurology of the medical school of the University of Pennsylvania, a few words might be said of the teaching of neurology and psychiatry in this University prior to the organization of a neurological department in 1871. Before this time lectures on diseases of the nervous system were by no means unknown in the history of the medical school. These were given by the professor of the theory and practice of medicine, and when it is remembered that this position was filled by men of such eminence as Chapman, George B. Wood, the first William Pepper and Alfred Stillé, it will be recognized that the instruction must have been the best of its kind for the periods in which these men taught. Much attention was also given by teachers of physiology like Jackson and Francis Gurney Smith to nervous phenomena and their relations to disease. In addition to the clinical and other teaching of neurology by those especially assigned for this purpose, it has continued to be the custom in the University for the professor of medicine to give some didactic instruction on the subject of nervous diseases.

The chapters on diseases of the nervous system in the work on the practice of medicine by Dr. George B. Wood are examples of careful and lucid presentation. Dr. Alfred Stillé contributed to our knowledge of diseases of the nervous system as a result of his personal investigations, especially notable being his monograph on epidemic cerebrospinal meningitis. A comparison of the two hundred and thirty-one pages in which Dr. Wood discusses nervous disorders in the 1847 edition of his text-book, with recent treatises on neurology like those of Gowers and Oppenheim, each of these comprising two large volumes, shows the remarkable increase in the number of separate affections now receiving consideration, and the extraordinary advances which have been made in our knowledge of these affections.

Lectures on the faculties and diseases of the mind formed a regular portion of the course of instruction given by Rush, the first in physiology and the second in physic or medicine. This is evident not only from the published syllabuses of his lectures on physiology and medicine, but also from his "Medical Inquiries and Observations upon the Diseases of the Mind," which represented in large part his teaching in the University of Pennsylvania. In addition to his instruction on diseases of the mind, Rush also taught a little neurology in its more general sense, as shown by a study of his works.

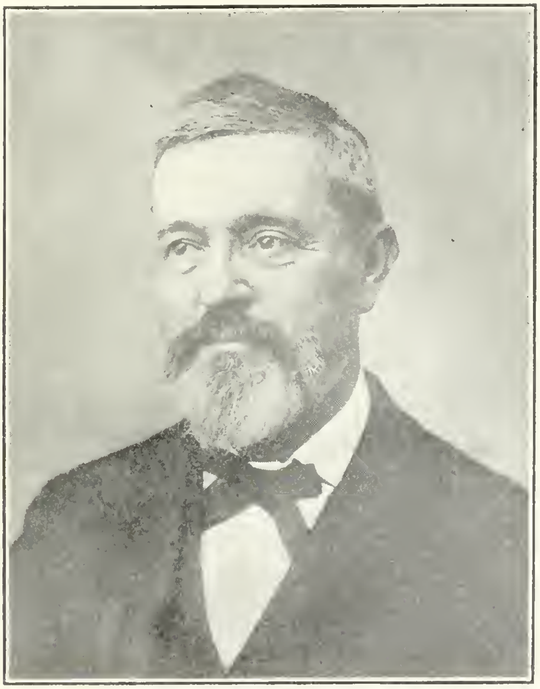
The Founding of the Present Neurological Department.

Specialism in medicine had its beginnings a few years before the founding of the neurological department of the University of Pennsylvania, and neurology, under the inspiration of the work of men like Hughlings Jackson, Hitzig, Ferrier and Mitchell, was demanding a separate place in medical teaching. In 1871 Dr. Horatio C. Wood was appointed lecturer on nervous diseases, and with characteristic zeal began the clinical teaching of neurology in the old medical hall on Ninth street above Chestnut. In this building he organized an outdoor or dispensary service for the study and treatment of patients suffering from diseases of the nervous system. This first dispensary for nervous diseases was under the charge of Dr. Morris Longstreth as chief assistant or chief of clinic. The service was conducted under the amphitheatre of the chemistry lecture room, on one side of the corridor which ran from the main entrance hall toward the room of Dr. Robert E. Rogers, professor of chemistry. About the same time the medical dispensary was started under the directorship of Dr. Roland G. Curtin, under the amphitheatre on the opposite side of the hall of entrance.

During the period between leaving this time-honored spot and going to the present campus in West Philadelphia, the teaching and the dispensary service were conducted in the building of the Philadelphia Medical College, which then stood on Ninth street near Locust. The medical buildings in West Philadelphia were not completed by the time when the contract with the government required that the old campus should be abandoned for the work on the new post office.

Dr. Longstreth continued in charge of the outdoor service during 1871 and 1872, and possibly part of 1873, when Dr. Louis Starr was made chief of clinic, and continued in this position during 1873 and part of 1874.

When the hospital was opened at its present position on the campus in West Philadelphia on June 4, 1874, Dr. Starr remained for a short time in charge of the dispensary service, when he was succeeded by Dr. Charles K. Mills as chief of clinic. Some records of the work done in the department by Dr. Wood and Dr. Starr, are contained in several articles published in the "Medical Times" of Philadelphia, in 1873 and 1874, under notes of hospital practice.



HORATIO C. WOOD.

History and Personnel of the Outdoor or Dispensary Service Since 1874.

Dr. Mills continued to hold the position of chief of clinic from 1874 to 1883 when he was appointed professor of diseases of the mind and nervous system in the Philadelphia Polyclinic and College for Graduates in Medicine. During his incumbency as chief of clinic several assistants to the service held appointments for varying periods. The first of these were Drs. George J. Clunas, William B. Christine and Dowling Benjamin. Dr. Edward T. Reichert, the distinguished professor of physiology in the medical school of the University, was an assistant physician in 1879-1880. Dr. Robert Glasgow served during the sessions of 1880-81, 1881-82 and 1882-83. Dr. James Hendrie Lloyd, who has since attained such eminence as a neurologist, medical jurist and writer, became an assistant in 1881 and continued for several years.

Dr. F. X. Dercum became chief of clinic in 1883, remaining in this position until his appointment in 1892 as clinical professor of nervous diseases in the Jefferson Medical College. His list of assistants, in addition to Dr. Lloyd, included Drs. I. Pearson Willits, R. P. Crandall, A. J. Plumer, Charles S. Potts, Joseph Sailer, William Evans, H. A. Tomlinson and Thompson S. Westcott.

Dr. Charles S. Potts succeeded Dr. Dercum in 1892, and gave long and valuable service in the position as chief of clinic from that date until he became professor of neurology in the Medico-Chirurgical College in the spring of 1907. From 1892 until 1901 he served with Dr. Wood, who held the chair of clinical professor of nervous diseases until the latter date. During the remainder of Dr. Potts' period of office he served with Dr. Mills and Dr. Spiller. Among those who acted as assistants to the dispensary service from 1892 to 1901 were Drs. William Evans, S. S. Kneass, Richard Wilson, H. A. Newbold, Walter W. Naylor and Eugene Lindauer. Between 1901 and 1907 the list of assistant physicians included besides Drs. Evans, Newbold, Naylor, and Lindauer, Drs. J. W. McConnell, D. J. McCarthy, T. H. Weisenburg, William T. Cummins, Horace Carncross, Carl D. Camp, T. J. Orbison, A. N. Baggs, A. R. Allen, E. J. Stein and Samuel Leopold. The tenure of office of these different assistants varied considerably. Their names are given in the present summary in the order of their appointment. Dr. D. J. McCarthy became professor of medical jurisprudence on the George B. Wood Foundation in 1904, and is widely known for his work in clinico-pathological investigations in neurology and tuberculosis. He is now the physician in charge of the large neurological service of St. Agnes Hospital, Philadelphia. Dr. T. H. Weisenburg was elected professor of neuropathology and associate professor of neurology in the Medico-Chirurgical College in 1907. Dr. Carl D. Camp was made clinical professor of diseases of the nervous system in the medical school of the University of Michigan, in 1907.

In 1907 Dr. J. W. McConnell became the chief physician to the dispensary. The next year the service was reorganized so that two sets of physicians should take charge of the outdoor service, each serving regularly on alternate days. Dr. J. W. McConnell and Dr. Alfred R. Allen became the chiefs of clinic, and the assistant physicians under this reorganization were Drs. H. A. Newbold, A. N. Baggs, S. Leopold, N. S. Yawger, Andrew H. Woods, John J. Repp and Paul H. Bickle. The next year the same staff continued and in 1910, the additional appointments of Drs. Frank B. Baird and Edward Mercur Williams were made. In 1911 Dr. Williams B. Cadwalader was added to the staff of assistant physicians.

The efficient and faithful service performed by the members of the dispensary staff, chiefs of clinic and assistants, cannot be over-estimated. To them has been largely due the reputation which the service has attained for good work in behalf of those who have applied for examination and treatment, and also for the supply of valuable material for neurological teaching purposes which has been constantly furnished, both for the clinical lectures and the instruction of special classes. As will appear later many of the members of the outdoor service have served in various teaching positions as lecturers, associates, instructors and assistant instructors.

The number of patients treated in the nervous dispensary has steadily increased from the foundation of the department. This increase, however, being large during recent years, since it has been better housed and has had a more thorough organization. The case books of the dispensary have been kept since September, 1872. The number of new cases applying to the dispensary in September, October, November and December of this year was 34; the number of new cases in 1873 was 80 and in 1874, 85. At this early period the number of return visits was not recorded. The attendance at the dispensary steadily increased, with occasional stationary periods. In recent years the hospital, like the University year, has begun September and ended August 31st. The number of new patients for the year ending August 31, 1909, was 619; the number of old cases (return visits) was 4,205; the total number of visits being 4824.

When the hospital of the University was opened in West Philadelphia in 1874, the dispensary for nervous diseases was first located in the most northern of the rooms used for outdoor service in the central wing of the original building. The accommodations consisted of two rooms; one about twice as large as the other, used as a waiting room for the patients, was entered by a door from the east. In the other, back of this, the patients were recorded, studied and treated so far as the use of electricity was concerned. The quarters were cramped and insufficient. In about 1894, when a considerable change was made in the arrangements for the dispensary services, three large rooms about the middle of the wing were set aside for the nervous dispensary, the door of entrance being now placed on the west side. These rooms were subdivided so as to make five in all. In the first room, which was large, the patients were received and separated, either for record or for treatment. The registration and care of old patients was assigned to one room, new patients being studied and recorded in the middle, larger room. The electrical treatment was carried out in two other rooms, each about one-half the size of the examination room. The furnishing and equipment of these rooms were much improved in 1904.

When the new dispensary building, erected on a part of the ground north of the line of the old hospital, was opened in 1909, its present excellent quarters were assigned to the outdoor neurological service. These consisted of four rooms beside the waiting room which is used partly by the neurological service and by the afternoon division of the medical service.

The patients receive their cards at a table in the waiting room and if new are sent for special examination and record to one of the two rooms set apart for this purpose. Returning patients are attended to by one of the assistant physicians to whom this duty is assigned. After a sufficient historical record has been made the new patient passes to a large room which is set apart for the examination of the case by the professor who is present or by the chief of clinic, the notes dictated by him being recorded by one of the assistant physicians in attendance. This room, which is supplied with benches, diagrams, blackboards, etc., is also sometimes used for teaching either post-graduate students or ward classes. The room is well lighted and is in every way admirably adapted for its purposes.

The electrical room is on the right of the hall of entrance. This room is now well supplied with the most improved forms of galvanic, faradic, and static apparatus. In addition to the improved apparatus for the use of galvanic and faradic currents, and the static machine, the service is supplied with one of the most modern forms of coil for currents of high frequency, and with an electric vibrator. These instruments, like the electric motor for the static machine, and one of the forms of cabinet apparatus used for galvanic and faradic applications, are run by the current derived from the electrical plant of the University.

All the apparatus and appliances are in good condition and have proved very serviceable.

The Indoor Neurological Service of the University Hospital.

Shortly after the present University Hospital buildings were opened, a men's ward for nervous diseases was established on the northern side of the Hospital on the first or main floor, opposite one of the long wards of the wing extending to the south. This ward had a maximum accommodation for about twelve beds, but for many years it was not fully occupied. During this time women patients suffering from nervous diseases were admitted to beds in the wards of the general medical service, some of these being attended by the clinical professor of nervous diseases and his assistants, and others by the members of the staff of internal medicine. The entire number of patients under the charge of the neurological chiefs did not as a rule exceed ten or a dozen. In 1908 an additional ward was set apart for the reception of nervous patients, this being on the third floor and also on the northern front of the hospital. The men patients were removed to this upper ward, because it was in direct relation with the men's medical ward in the wing, while the ward on the first floor was given over to women patients, because the medical ward for women was opposite to it. Both of these wards were thoroughly furnished and equipped through the energetic work of the auxiliary committee for nervous wards of the Board of Women Visitors. Between eight and nine hundred dollars were raised. New beds, tables, desks, wheeling-chairs, screens, air-beds, water-beds, etc., were bought and the wards were put in excellent condition.

In these wards, ward or bedside teaching is given by the assistant instructors and the patients in them are much used by the professors for their clinical lectures. The total number of cases usually available in these wards is between fifteen and twenty. They supply, in conjunction with the excellent outdoor service, all the material necessary for the neurological teaching in the Hospital.

The work in the nervous dispensary and in the nervous wards of the University Hospital is far from fully indicating that done by the neurological staff, which acts in harmony with other departments in the investigation of important cases. This is particularly true with regard to the surgical, medical, ophthalmological, orthopaedic and children's services. Many cases are referred by the surgeons on duty, for examination to the neurologists, these including all forms of disease of the brain, spinal cord, and peripheral nerves, susceptible of improvement by surgical procedure. During the last ten years the number of cases of tumor of the brain which have been studied for surgical treatment has been very large; also particularly to be noted are cases of tic douloureux, in which Gasserian, sensory root, and peripheral operations have been performed: cases of spinal fracture, tumor, and pachymeningitis; cases of nerve stretching, section, and anastomosis, and posterior root operations for pain and spasticity. The neurological department is under special obligations to the professor of ophthalmology and his staff, for assistance in the investigation of cases sent to this department from the neurological service, and also for the reference of cases from the ophthalmological service. The interchange of work and counsel between the medical and the neurological departments has gradually increased, with advantage to both services.

The Nervous Wards of the Philadelphia General Hospital and Their Use by the Neurological Department of the University.

In writing the history of the department of neurology of the University of Pennsylvania, it is necessary to take into account the large part played by the nervous wards of the Philadelphia Hospital in teaching and in investigation. These wards for men and women constituting the neurological department of the Hospital, were founded by Dr. Mills in 1877 with about a score of patients. The number increased at first slowly, and later with considerable rapidity, as additions were made to the accommodations, first by taking the unused wooden pavilions, later through the erection of brick pavilions, and still later in 1907 by the removal of the men's nervous wards to a portion of the old out-wards in the eastern wing of the original quadrangle, and the women's wards to the western wing of the quadrangle. In 1908 the women's wards were again removed to the brick pavilions in the area outside of the eastern confines of the quadrangle, where they have since remained.

In 1878 the number of patients in the nervous wards of the Philadelphia Hospital was 35. In 1910, including now the psychopathic wards established in 1906, the number had reached the total of about 400 patients.

From a very early period after the organization of the nervous wards of the Philadelphia Hospital, they were used for the purpose of teaching students of the medical school of the University, and of other medical institutions. The teaching at first was twofold: clinical lectures delivered in the old medical clinical hours, from 10 to 11 on Wednesdays and Saturdays, and secondly, somewhat irregular ward instruction given to small classes at the bedside of the patients, or in the rooms adjoining the wards.

It will not serve any useful purpose to review year by year the instruction which was given in the clinical amphitheatre to both the third and fourth year classes of the University and to ward and post-graduate classes. It is sufficient to say that the Hospital has been utilized not only for amphitheatre instruction in nervous diseases but also for undergraduate ward classes and post-graduate teaching in neurology. As most of the patients in the Philadelphia General Hospital, which is the city hospital, remain in the wards for months, or even years, and as almost all forms of organic disease of the nervous system are illustrated in the patients remaining, it is easy to classify the diseases taught and in this way give almost as systematic instruction as in a purely didactic course of lectures.

Most of the members of the neurological teaching staff of the University are connected officially with the Philadelphia General Hospital either as neurologists, assistant neurologists or registrars, these positions giving official access to the material under the rules of the Hospital. The Philadelphia General Hospital, the medical hall or laboratories building of the University and the hospital of the University are situated in close proximity to each other, this enabling lectures and ward classes to be rostered in either of these buildings in such a way that the students can pass from the University campus to the city grounds, or the reverse, in successive hours.

For some years the instruction of University classes in neurology in the nervous wards of the Philadelphia Hospital has not materially changed, but has become more systematic and thorough with the growing experience of the professorial staff and instructors. Opportunities are sometimes given to students to conduct particular investigations in clinical neurology or in neuropathology under the supervision of the attending physicians who are connected with the teaching staff of the department of neurology of the University. Occasionally valuable contributions have been made in this way by students in the year before their graduation.

The Organization and Growth of the Neurological Teaching Staff.

In 1871, as already stated, Dr. H. C. Wood was elected by the Trustees of the University, lecturer on nervous diseases. In 1874 or 1875, Dr. Wood's lectureship was made a clinical professorship and he continued as clinical professor of nervous diseases until 1901, when he was succeeded by Dr. Mills as clinical professor of nervous diseases, Dr. Spiller being at the same time appointed assistant clinical professor, and in 1903 the titles of these chairs were changed respectively to professor of neurology and associate professor of neurology, and in the same year Dr. Spiller was appointed professor of neuropathology.

A continuous line of instructors below the rank of professor has co-operated for many years with the professorial staff in the teaching of neurology. The first position created was that of lecturer on electrotherapeutics, which was filled by Dr. Mills in the session of 1877-78 for the first time. The instruction was given in the spring course. Dr. Mills continued to hold this position until the close of the session of 1880-81, when the title was changed to lecturer on mental diseases and electrotherapeutics, continuing as such during the sessions of 1881-82 and 1882-83. In 1883 the position of Dr. Mills was again changed to that of lecturer on mental diseases, which position he held continuously until 1892, when he was made lecturer on nervous and mental diseases. From this time until his election to the chair of clinical professor of nervous diseases he continued to lecture during one-half of the session on nervous diseases at the Philadelphia Hospital by special arrangement with Dr. H. C. Wood, at the same time giving his courses on mental diseases and medical jurisprudence. Dr. Wood, at this time not being a member of the staff of the Philadelphia Hospital, was desirous that the students should have the opportunity of receiving instruction from the abundant neurological material especially illustrating organic diseases at the Philadelphia Hospital. During a portion of the period between 1877 and 1892 or 1893, ward class instruction in the Philadelphia Hospital was at times given by Dr. Mills to members first of the elective and afterwards of the compulsory fourth year course, and also to occasional post-graduate students, who were not, however, registered regularly as such at the University as the official post-graduate course was not continued from 1885 to 1904.

The first to hold the official title of instructor in nervous diseases in the neurological department was Dr. Francis X. Dercum, in 1883-84. Dr. Dercum continued in this position until his election to the position already indicated, in the Jefferson Medical College in 1892. Dr. James Hendrie Lloyd was made instructor in electrotherapeutics in the session of 1883-84, remaining in this position until the close of the session 1889-90, when he was succeeded by Dr. Charles S. Potts. The position of Dr. Potts was expanded to that of instructor in electrotherapeutics and nervous diseases for the session of 1892-93, and he continued to hold this title until the close of the session of 1902-03.

On the reorganization of the staff in the session of 1903-04, Dr. J. W. McConnell and Dr. T. H. Weisenburg were made instructors in neurology, Dr. Potts becoming associate in neurology. Dr. Eugene Lindauer and Dr. Carl D. Camp were made instructors in electrotherapeutics in 1906-07. During the sessions of 1904-05, 1905-06, and 1906-07, Dr. Weisenburg was instructor in neurology and neuropathology.

Since 1904-05 the following have been included in the staff of associates and instructors: Associates, Drs. Charles S. Potts, J. W. McConnell and A. R. Allen; instructors in neurology, Drs. J. W. McConnell and T. H. Weisenburg; instructors in neurology and neuropathology, Drs. T. H. Weisenburg, S. D. W. Ludlum and A. R. Allen; instructor in electrotherapeutics, Dr. Eugene Lindauer; instructor in electrotherapeutics and neuropathology, Dr. C. D. Camp. In the session of 1908-09 Dr. Alfred R. Allen was made lecturer in neurological electrotherapeutics. Dr. Andrew H. Woods was made lecturer on electrotherapeutics in 1911.

Since the session of 1908-09 Drs. Samuel Leopold, N. S. Yawger, and Williams B. Cadwalader have been assistant instructors in neurology.

The assistant neurologists to the hospital since 1883 have been Dr. Francis X. Dercum (1883-93), Dr. Charles S. Potts (1893-1907), Dr. J. W. McConnell (1905-11), Dr. A. R. Allen (1909-11), and Dr. Edward M. Williams (1910-11).

In 1902 Dr. Eugene Lindauer was made registrar to the professor of neurology and was succeeded in this position in 1906 by Dr. S. D. W. Ludlum.

Present Organization of the Neurological Staff.

Charles K. Mills, M.D., Professor of neurology.

William G. Spiller, M.D., Professor of Neuropathology and associate Professor of neurology.

J. W. McConnell, M.D., Associate in neurology and instructor in neurology.

Alfred R. Allen, M.D., Associate in neurology and instructor in neuropathology.

S. D. W. Ludlum, M.D., Instructor in neurology and in neuropathology.

Andrew H. Woods, M.D., Lecturer on electrotherapeutics.

Samuel Leopold, M.D., Assistant instructor in neurology and assistant in neuropathology.

N. S. Yawger, M.D., Assistant instructor in neurology.

Williams B. Cadwalader, M.D., Assistant instructor in neurology and assistant in neuropathology.

Edward Mercur Williams, M.D., Assistant in neuropathology.

HOSPITAL AND DISPENSARY STAFF.

J. W. McConnell, M.D., Chief of clinic and assistant neurologist.

Alfred R. Allen, M.D., Chief of clinic and assistant neurologist.

Edward Mercur Williams, M.D., Assistant neurologist.

S. D. W. Ludlum, M.D., Registrar.

ASSISTANT PHYSICIANS TO THE DISPENSARY.

Henry A. Newbold, M.D.
Samuel Leopold, M.D.
N. S. Yawger, M.D.
Andrew H. Woods, M.D.
John J. Repp, M.D.
Frank B. Baird, M.D.
Edward Mercur Williams, M.D.
Williams B. Cadwalader, M.D.

Post-Graduate Instruction in Neurology.

The first post-graduate department in the medical school of the University of Pennsylvania was established in 1882. The instruction, which was on nervous diseases and electrotherapeutics, was given in the spring and summer course in the session of 1882-83, by Dr. Wood and Dr. Mills. When Dr. Mills resigned from his position as chief physician to the dispensary to go to the Philadelphia Polyclinic, the post-graduate instruction was continued by Dr. Wood and by Dr. Wharton Sinkler at the Orthopaedic Hospital and Infirmary for Nervous Diseases. The post-graduate teaching in the session of 1884-85 was conducted by Dr. Francis X. Dercum and Dr. James Hendrie Lloyd.

The University post-gradute department was discontinued at the close of the session of 1884-85. A new post-graduate school was opened during the session of 1904-05, the staff then conducting the neurological teaching being Dr. Mills, who gave instruction both in the University Hospital and in the Philadelphia General Hospital, and Dr. Potts and the assistant physicians in the dispensary whose course included electrotherapeutics as well as nervous diseases. A post-graduate laboratory course in neurology was given by Dr. Spiller. Dr. Charles W. Burr, in addition to his clinical lectures and demonstrations in psychiatry, had a post-graduate course in neurology at the Philadelphia General Hospital. During the next two sessions post-graduate instruction was given by the same staff, but the course was discontinued at the close of the session of 1906-07.

The classes in attendance were small but attentive to the work, both as given in the hospitals and in the laboratory of neuropathology.

Independently of any general post-graduate department in the University, graduates in medicine from time to time have taken up special work in the department of neurology. These men have as a rule proved to be desirable students and in some instances have been made temporarily voluntary assistants both to their own advantage and to that of the clinical work. The manner in which this post-graduate work is regarded by those receiving it can perhaps be estimated by a quotation from an article recently published in "The Journal" of the Missouri State Medical Association, August, 1910, by Dr. R. E. Castelaw, of Kansas City, Mo. In this article the general subject of post-graduate teaching of neurology in America is discussed and referring to the work done at the University of Pennsylvania the writer speaks as follows:

"I feel I am safe in saying that there is no place in America where neurological material may be found in such great abundance and variety, and so well classified, as in Blockley, and I doubt if there is a place in the world its superior in this respect.

"During the recent Christmas holidays (1909) I tabulated the cases." (This tabulation included 400 cases, mostly organic.) * * * *

"During my attendance as a post-graduate student at the hospital of the University of Pennsylvania and at the Philadelphia General Hospital I had the opportunity of observing operations covering almost the entire field of neurological surgery. These included radical and decompression operations for brain tumor; operations for the removal of tumors of the spinal cord and the evacuation of spinal cysts; for circumscribed serous meningitis; nerve sections for various purposes; sections of the posterior spinal roots for the relief of spasticity; operations for nerve anastomoses; peripheral and sensory root sections of the fifth nerve, and extirpation operations on the Gasserian ganglion.

"Much of the operative work observed by me was done by Dr. Charles H. Frazier, Dr. Edward Martin, and others connected with the University staff.

"Post-graduate students sometimes have the opportunity of becoming voluntary assistants, taking regular part in the recording and examination of the cases. Great attention is paid to the manner in which the cases are recorded, as regards not only the history of the case, but also the systematic record of motor, sensory, reflex, visceral, and other symptoms.

"The outdoor service of the University Hospital is provided with a thorough electrical equipment and instruction in electro-diagnosis and electrotherapeutics is given in courses of five or six weeks.

"In connection not only with the neurological teaching, but in the entire work of the neurological department at the University of Pennsylvania, the professors in charge, Drs. Mills and Spiller, take a continuous active interest, attending the dispensary service regularly several times a week, visiting the neurological wards of the hospital almost daily during their terms of service, and also visiting the wards of the Philadelphia General Hospital four or five times weekly. In the different hospital services some practical instruction is given by the heads of the departments at least four or five times a week, this being supplemented and enlarged by the instructors and assistants in neurology."

Present Methods of Teaching in the Neurological Department.

The instruction in neurology during the fourth year is almost entirely clinical, although an effort is made to systematize the work and demonstrate neurological principles in connection with the cases shown.

In the first place the course consists of clinical lectures and demonstrations in the University Hospital by the professor and associate professor. One lecture a week is given before February 1st by Dr. Mills, and after February 1st by Dr. Spiller. Secondly, one clinical lecture a week is given in the Philadelphia General Hospital before December 1st, in the first half of the period by Dr. Mills and in the second half by Dr. Spiller. Thirdly, ward class or section instruction is given before February 1st, in the Philadelphia General Hospital and in the nervous dispensary of the University Hospital by the instructors in neurology, Drs. J. W. McConnell, A. R. Allen, and S. D. W. Ludlum. Fourthly, instruction in the form of ward visits is given once weekly to small sections in the wards of the University Hospital by assistant instructors.

The material for the clinical lectures on neurology at the University Hospital is drawn from the indoor neurological service of the hospital and from the outdoor or nervous dispensary service. The latter is a fairly large and growing service and furnishes most forms of walking cases of nervous disease. The indoor service has already been sufficiently described. While not large the material of the indoor service is usually valuable for teaching, as the cases are whenever possible chosen with this end in view.

The professors of neurology keep a careful list of all the subjects upon which they lecture, in order that these may be reviewed at the end of the year. A study of this list shows that the subjects cover a wide range and complement the systematic instruction on chronic organic disease given at the Philadelphia General Hospital.

Clinical lectures largely dependent upon an outdoor service must necessarily be somewhat unsystematic, the order in which the diseases are lectured upon being dependent to a considerable degree upon the time when cases of certain forms of disease appear. Whenever possible outdoor and indoor cases of the same group or allied groups are presented to the class in the same or in successive lectures. The number of diseases actually discussed at the clinical lectures is not fully represented by the cases shown, as close attention is paid in the lectures to the differentiation of these cases from others from which they need to be diagnosticated.

Especial attention is given in the lectures to clinical history, diagnosis, and prognosis, although the pathology of the affections shown is discussed to some extent, and the cases, both at the Philadelphia General Hospital and at the University, are used to illustrate when possible the course on neuropathology given in the third year. The treatment of nervous diseases is discussed to some extent in the different lectures in connection with the particular cases shown. In addition several special lectures are given each year on the practical therapeutics of nervous diseases. This instruction, which is given toward the end of one of the sessions, is made to apply largely to the diseases and conditions which have been illustrated in the lectures of the term, serving in this way as a therapeutic summary or review. Special measures of treatment, such as rest, massage, movements, exercises and hydrotherapeutics, are briefly described.

Small sections of the class are given instruction, usually each for about five weeks, in electrodiagnosis and electrotherapeutics by the lecturer on electrotherapeutics. In this course instruction is given in the reactions of degeneration, an effort being made to show how these differ from the normal reactions, what their presence signifies, and the method of detecting their presence. The methods of using both the galvanic and the faradic currents in the treatment of such affections as motor paralysis, neuralgia, neuritis, and other diseases in which these currents are applicable are taught; also the method of giving general faradization. The class receives some instruction in the nature and indications for the use of the sinusoidal current; also in the methods of employing static electricity in the treatment of neurasthenia, hysteria, and other neuroses, anemia and chronic neuritis. The students have considerable opportunity to do the work in electrodiagnosis and electrotherapeutics themselves.

I have already spoken of the facilities offered to the students of the University by the nervous wards of the Philadelphia General Hospital, and in part of the methods there adopted.

The entire fourth year class is divided into nine sections of about thirteen to fifteen men each. Each of these sections is met continuously for five weeks, the instructors giving one hour three days a week for the first half of the session. Particular attention is paid to the methods of investigating the cases and eliciting the symptoms, each student being given as much opportunity as possible to do the work of examining for himself. The students are expected to make diagnoses, these being discussed by the other members of the class and the instructors. The methods of interpreting symptoms and conditions are discussed.

The instruction given in these ward classes at the Philadelphia General Hospital, owing to the ability and earnestness of the instructors and to the large amount of material always at their command, is in the highest degree valuable. Probably in no other institution in this country are equal opportunities for the study of the symptomatology, diagnosis and pathology of organic nervous diseases offered. The students as a rule are highly appreciative of these opportunities. The attendance upon the lectures and ward classes is good, and close attention is paid to the teaching.

Elective instruction in neurology is sometimes given during the second half of the session to such students as have the time and the wish to avail themselves of the opportunity.

The Laboratory of Neuropathology and Neuropathological Investigation and Teaching.

The department of neuropathology was established in 1900-1901 when Dr. Spiller was appointed demonstrator of neuropathology. The course was optional at first. During the first year instruction was given to a number of undergraduate students and to several physicians. In 1901 Dr. Spiller was appointed assistant professor of neuropathology and assistant professor of nervous diseases. When the new medical laboratories were opened in 1904 the laboratory of neuropathology was established, and the course in neuropathology was given in the new building. Opportunity was offered to physicians to undertake original investigation, and in the years that have passed since 1904 a considerable number have availed themselves of the conveniences afforded. A few undergraduate students have done original work in the department, and have prepared and published creditable papers embodying the results of their researches.

The course, after it was established in the new laboratory and made obligatory for all the third year class, was arranged for the demonstration of gross specimens and for explanatory lantern lectures with microscopical slides. In 1903 Dr. Spiller was appointed professor of neuropathology and associate professor of neurology and these positions he holds at present.

It has been the purpose of the course in neuropathology to prepare the third year students for their clinical work in the neurology of the fourth year, and to correlate their studies on anatomy, physiology and pathology with the clinical study of nervous disease. The course has never been one devoted solely to the pathology of the nervous system, but has been planned on broader lines. Attention is directed to cerebral and spinal localization, to the symptoms likely to be caused by the various lesions according to their location in different parts of the brain or spinal cord, to the functions of the various tracts and groups of nerve cells, to the methods of determining the various levels of the spinal cord by counting the segments, after the spinal cord has been removed from the cadaver, to the recognition of the gross appearances of cerebral and spinal lesions, to the methods of preparing and staining the nervous tissues, etc. In this way considerable stress is laid on the anatomy, physiology and pathology of the nervous system. In the lectures with lantern demonstrations the various systemic and diffuse degenerations are exhibited and used to explain the symptoms of those diseases whose pathology must be studied by the microscope. The loss of tendon reflexes and of control of the bladder, in tabes dorsalis; the existence of spasticity, of exaggerated reflexes and of weakness in amyotrophic lateral sclerosis, for example, are explained by the lesions demonstrated.

In this way the students are prepared for the clinical neurology of the fourth year, and enter upon this somewhat difficult study with an understanding that could not be obtained without the instruction given in the third year.

The course at first extended through the greater part of the year, but it was later thought best to limit it to the first half of the session. During the months of October and November the third year students are instructed for an hour and a half three times a week, by the professor of neuropathology and his assistants, the class being divided into three sections containing about forty to forty-five men each. During December and January a lantern lecture is given to the entire class once a week by the professor of neuropathology.

A number of gentlemen have served in the capacity of instructors or assistants during the period the course has been in existence. Of these Dr. Carl D. Camp and Dr. T. H. Weisenburg, as noted elsewhere, have been called to important professorial positions in other medical institutions. The instructors and assistants at present connected with the department are Dr. S. D. W. Ludlum, Dr. Alfred R. Allen, Dr. Samuel Leopold, Dr. Williams B. Cadwalader and Dr. Edward Mercur Williams.

Those who at present are engaged in original investigations in the laboratory are Drs. S. D. W. Ludlum, A. R. Allen, S. Leopold, J. H. W. Rhein, W. B. Cadwalader, A. H. Woods, E. M. Williams, N. S.

Yawger, H. Carncross, and E. Corson White. A large amount of work has been accomplished by these investigators, and the results of their labors have been made known in the contributions in the five volumes, to which reference will be made.

The specimens collected in the laboratory now number nearly six hundred, and those suitable for gross exhibition have been placed in cabinets, covering two of the walls of the laboratory, where they are exposed to the benefit of all desiring to make a study of the gross pathology of the nervous system.

The laboratory has been fortunate in having the services of Drs. E. Corson White and S. D. W. Ludlum in carrying out the technique of the Wassermann and Noguchi reactions, and about six thousand examinations have been made to date.

The Teaching of Psychiatry at the University.

After the time of Rush, psychiatry was not formally taught at the University of Pennsylvania until the appointment of Dr. Mills as lecturer on mental diseases in 1881, a position which he held until 1893 when he became professor of mental diseases and of medical jurisprudence. He was succeeded in these positions by Dr. Charles W. Burr, who became the professor of mental diseases in 1901 and by Dr. D. J. McCarthy, who was appointed professor of medical jurisprudence on the George B. Wood Foundation in 1905. The teaching of Dr. Burr is chiefly conducted at the Philadelphia General Hospital, his cases being furnished by the insane department of this institution. The instruction is of unusual value, because not only of the ability of the occupant of the chair, but because of the abundance of material which is not surpassed by that supplied to any other medical teaching institution.

Contributions To Neurological Literature.

From the time Dr. Horatio C. Wood was appointed lecturer on nervous diseases in the University many of the members of the neurological staff have been active contributors to neurological literature. Dr. Wood, himself, early in his career as lecturer and clinical professor of nervous diseases, published a number of valuable papers in the domain of clinical neurology. In his great work on Physiological Therapeutics, and in numerous contributions in current literature, his chapters or articles on drugs especially influencing the nervous system are of unusual merit. His studies on fever, published as a Smithsonian contribution in 1880, dealt in large degree with the functions of the central nervous system. In 1887 he published a work under the title of "Nervous Diseases and Their Diagnosis," and his monograph on nervous syphilis was a valuable contribution to this subject, and one of the first of its kind appearing in this country. It was largely based upon clinicopathological work done during his connection with the Philadelphia Hospital. While the greatest achievements of this leading exponent of American medicine will always be recognized as exhibited in his work on physiological therapeutics, he has left an enduring reputation both as a neurological teacher and writer.

Dr. Francis X. Dercum, while still connected with the University of Pennsylvania as chief of the nervous clinic and as instructor in nervous diseases, contributed many of the articles and monographs through which he has attained his present high place in neurology.

The contributions of Dr. Charles S. Potts, now professor of neurology in the Medico-Chirurgical College, of Dr. T. H. Weisenburg, associate professor of neurology and professor of neuropathology in the same institution, of Dr. Carl D. Camp, professor of nervous diseases in the University of Michigan—all of whom as has been shown, did valuable service in connection with the neurological department, need not be here reviewed. Much of their published work was done while they still were connected with the University of Pennsylvania.

Consultation of such medical periodicals as the "Journal of Nervous and Mental Disease," the "American Journal of Insanity," the "American Journal of the Medical Sciences," the Journal of the American Medical Association," "Brain," the English Journal of Neurology; the "Review of Neurology and Psychiatry," edited by Bruce, and many other journals, will afford evidence of the industry and ability of those holding positions in the neurological department of the University below the rank of professor.

The indebtedness of the medical profession, and especially of neurology, to Dr. William Gibson Spiller, can not be over-estimated. Prior to 1904, as indicated by the writer in his history of Philadelphia neurology, Dr. Spiller's publications on neurological subjects had reached the number of eighty, many of these being of unusual value, and in the few years which have elapsed since then, he has contributed an even larger number. In association with Dr. William Campbell Posey he edited a work on the "Eye and the Nervous System," which has passed through two editions in less than three years.

Dr. Mills during his long connection with the University of Pennsylvania has published numerous articles and monographs, chiefly neurological, a textbook on the nursing of the nervous and the insane and a volume of about one thousand pages on diseases of the brain and cranial nerves.

Since 1905 five volumes have been issued from the department of neurology and the laboratory of neuropathology, these volumes in the main constituted by the collection and binding of reprints or papers published in current neurological and general medical literature. An examination of these volumes will show that the work contributed has been done not only by the professors, but also by the instructors and assistants and in a few instances by undergraduate and post-graduate students who have conducted investigations under the supervision of the professor of neuropathology. In the pursuit of the work which has resulted in these contributions great opportunities have been afforded through the connection of the members of the University staff with the neurological service of the Philadelphia General Hospital. Cases can here be followed for years and many necropsies are obtained, the specimens as a rule going to the University laboratory of neuropathology for careful investigation. The members of the clinical and of the neuropathological staff work together in forwarding such investigations. Histories are taken by the visiting neurologists and their assistants, and the clinical facts thus obtained are correlated by the pathologists to the lesions found.

It is worthy of note, as showing the interest taken by undergraduates in neurology, that four of the eleven papers, or about 36 per cent., presented at the first annual meeting of the undergraduate Medical Association of the University of Pennsylvania in 1908, were on neurological subjects.

EPIDURAL ASCENDING SPINAL PARALYSIS.

By WILLIAM G. SPILLER, M.D.,
Professor of Neuropathology in the University of Pennsylvania.

(With Plate 29)

Ascending spinal paralysis has been recognised many years, but so far as I know no case has been reported in which this paralysis was caused by extensive epidural lesions, nor has any reference been made to such a form of paralysis. It is important to recognise that a lesion external to the dura, and extending from the lumbar to the cervical region, may be the cause of ascending paralysis.

In those cases in which the degeneration of the lateral columns is intense, as in the first case of this paper, the paralysis may be spastic in the lower limbs, even though the lumbar roots be somewhat implicated. In other instances when the degeneration of the lateral columns is slight, as in the second case, the paralysis of the lower limbs may be flaccid from the implication of the lumbar roots.

The clinical diagnosis between intradural and extradural ascending paralysis may be very difficult or even impossible, but as the latter form is of rare occurrence, a mistake is not frequently made. Ankle clonus with loss of patellar reflexes, as in the second case, would imply that the lesion is in the lumbar region and above the reflex arc for the Achilles tendon reflex, but it would not justify a diagnosis of extradural from intradural lesion ; it is even questionable whether this phenomenon would be more likely to result from an extradural lesion. Motor palsy without sensory symptoms would be more indicative of an intramedullary than of an epidural lesion.

The first case of this type that has come under my observation was seen in 1902, although its importance in this respect was not recognised at that time. In that year Dr Charles K. Mills and I reported a case of external spinal pachymeningitis, but ascending paralysis was not considered.[1] The history of the case was incomplete. The man was 42 years old. He had been blind since he was three years of age. Numbness began in the right great toe in June 1895, and gradually spread to the knee, and then attacked the other foot and leg in the same way. He had no pain. Previous to the onset of the numbness he had had swelling and tenderness in the legs and pain in the lumbar region. He could walk until November 1895, but his knees became bent. He began to have nocturnal incontinence of urine in July 1895. The tendon reflexes became exaggerated and spasticity developed in the lower limbs. In an examination made, 12th October 1898, sensation was found to be intact; later it was stated that the man could extend his lower limbs and stand, but could not walk, and that he was very spastic in the lower limbs. Nothing was recorded as regards the condition of the upper limbs beyond the occurrence of gangrene in the right hand.

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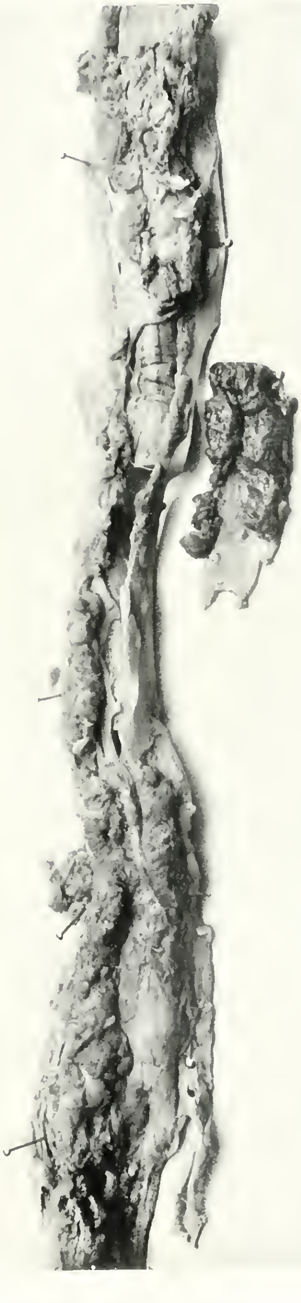
[1] Mills and Spiller, *Brain*, 1902, p. 318.

The external surface of the spinal dura on the ventral aspect was found firmly adherent to the bodies of the vertebræ throughout the spinal canal. Some of the nerve cells of the anterior horns of the lower cervical region appeared atrophied, and here and there a nerve cell was found with displaced nucleus. The columns of Goll in the cervical region were much degenerated. The ventral and dorsal roots of the cervical region appeared to be normal, but there is doubt concerning their complete integrity. As regards the crossed pyramidal tracts, it was stated that these tracts in the cervical region contained a slight excess of neuroglia, but by Weigert's hematoxylin stain did not appear degenerated. This statement from further examination seems inaccurate. The crossed pyramidal tracts in the lower cervical region were distinctly though slightly degenerated by this method of staining.

The degeneration was very intense in the peripheral portions of the cord in the mid-thoracic region.

The nerve cells of the anterior horns of the lumbar region were much like those of the cervical region. The crossed pyramidal tracts were degenerated. The posterior columns were partly degenerated. There was some question as regards the condition of the anterior roots throughout the cord.

One of the ulnar nerves examined showed much overgrowth of fibrous connective tissue, and considerable degeneration of nerve fibres. A similar but not quite so intense alteration was



Photograph of the spinal cord showing the tumour on the posterior aspect of the dura. The small piece represents a portion of the dura removed for preservation in Kaiserling's fluid, in order to retain the colour.

To illustrate Paper by Dr Spiller.

found in the musculo-spiral and median nerves. A section from one of the plantar nerves resembled that from the ulnar.

The case was reported as an extraordinary example of external spinal pachymeningitis, and such it was, but by regarding it in the light of the pathological findings, there can be little doubt that the upper limbs were affected. The case was not carefully studied during the last year or two of life. The spasticity and weakness began in the lower limbs, but the distinct though moderate degeneration of the crossed pyramidal tracts in the lower cervical region, with some alteration of the nerve cells of the anterior horns of the cervical region, probably caused a similar condition in the upper limbs. It is surprising that spasticity could have existed with so much overgrowth of fibrous tissue in certain of the nerves, especially in the peroneal and ulnar nerves, with considerable degeneration of nerve fibres, but the degeneration of the crossed pyramidal tracts, intense below the cervical region, explains the spasticity.

The condition of the nerves suggests the interstitial, hypertrophic, progressive neuritis of childhood of Dejerine and Sottas, but the symptoms were not those of this disease. There may have been a tendency to fibrous overgrowth in the nerves as well as on the external surface of the dura.

From the history and pathological findings the conclusion therefore is justified that this case was probably one of slowly ascending paralysis from epidural lesion. Numbness in the toes, extending to the knees, tenderness in the legs, and pain in the lumbar region, are suggestive of a lesion in the lumbo-sacral region, and would not be likely to occur from a lesion in the mid-thoracic region.

More demonstrable of epidural ascending spinal paralysis, though in an acute form, is the following case. The lesion was an epidural sarcoma with some resemblance to endothelioma.

The patient had been under the care of several physicians (Drs Hunt, Talley, and Musser) before he came into my service. The following notes were taken January 26 and 27, 1910 :—

The patient was a man 37 years of age. In October 1909 he had had pleuro-pneumonia, and had not been well since. He had lost 50 pounds since November 1909. In the early part of November he began to have pain in the back of the legs and in many of the muscles, and stiffness in the back. The pain gradually disappeared, but he became progressively weak, until, about a month before he entered the hospital, he was obliged to take to his bed.

He had very little power in the lower limbs, but could move the thighs slightly on the abdomen and move the ankles slightly; the movements, however, in all parts of the lower limbs were so feeble as to amount almost to complete palsy, and the lower limbs were flaccid. The patellar reflex was exceedingly weak on each side, but distinct and persistent ankle clonus was present on each side.

Touch of the finger, the prick of a pin, and hot and cold test tubes were promptly recognised in all parts of the lower limbs, but all forms of sensation were equally, though slightly, diminished here. Atrophy was not present anywhere. Babinski's upward movement of the toes was obtained on each side. The tonicity at the knees and hips was equally diminished, but was normal or more than normal at the ankles. The cremaster and abdominal reflexes were lost. The man was catheterised twice daily, and control of the bowels was lost. The lumbar and thoracic vertebræ were tender to pressure as far as the mid-thoracic region.

The grasp of the right hand was a little diminished, but that of the left hand was greatly impaired. The biceps tendon reflex was distinctly exaggerated on each side, especially on the left side. The tonicity in the left upper limb was somewhat diminished at the wrist and elbow, but was normal in the right upper limb. Sensations of pain, touch, temperature and position, and recognition of objects, were affected in the upper limbs only in so far that they were not quite so acute in the left upper limb. Ataxia in the finger to nose test, probably from weakness, was obtained on the left side but not on the right side. The cranial nerves were not affected. The Wassermann reaction made by Dr E. Corson White was negative.

By February 14, 1910, the left hand had become almost completely paralysed, and the weakness of the right hand had increased greatly. Edema of the lungs developed, and death occurred on this date. Permission was obtained only for removal of the spinal cord, and this was done by Dr Howard T. Karsner. On cutting through the muscles of the back, an abscess cavity, spherical, 2 cm. in diameter containing thick creamy pus, was encountered about the level of the tenth thoracic vertebra. Upon cutting through the laminæ, a mass of thick creamy pus was found extending throughout the length of the canal, moderately adherent to the dura, but without any evidence of penetration through the dura. The dura was opened in two places, and the subdural membranes were found to be smooth and but moderately congested. The spinal cord was given to me for examination. A sarcoma, resembling in the chain-like arrangement of the cells an endothelioma, was found on the posterior external aspect of the dura, extending from the sacral to the cervical region. Moderate secondary degeneration of the crossed pyramidal tracts and of the lateral periphery of the cord was found. Pus was not detected in the membranes by the microscope, and what was found in the spinal canal may have leaked in at the time of the necropsy; the relation of the pus, therefore, to the sarcoma is not easily determined. An extensive sarcoma, such as existed in this case, on the external surface of the dura seems to be an unique finding. The case could not be considered merely as one of tuberculous caries of the vertebræ, for the appearance of the masses of cells under the microscope was distinctly that of sarcoma. Whether a tumour had become infected, or whether a purulent process had arisen in the lungs and become associated with an independent tumour mass upon the dura, must remain undecided on account of the unavoidably incomplete necropsy.

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HEMICHOREA, HEMIATAXIA, HEMIPARESIS, AND DILATED PUPIL, PROBABLY DUE TO A LESION OF THE SUPERIOR CEREBELLAR PEDUNCLE*

By CHARLES K. MILLS, M.D.
PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA

Mrs. C., a widow, aged fifty-one years, was first seen by me on November 23, 1909, having been referred by Dr. M. S. Mengel, of Elverson, Pa., and Dr. John Wells, Chester Springs, Pa. About six months and a half before coming under observation she had a seizure which left her somewhat incapacitated or paralyzed on her right side, arm and leg. The attack came on while she was walking around. She first had an unnatural feeling in her right arm, then in her leg, and both members gradually got so that she could not use them properly. Speech was not affected. She was not unconscious at any time.

For several weeks previous to the apoplectiform seizure she had had at times a swimming or giddy sensation, and had also a little headache above the left eye, and ever since she has continued to have some pain in this region.

It was evident at first sight that her arm was the seat of a continuous tremor, which increased when she used it.† The tremor was of a moderately coarse type. The oscillating hand was held slightly flexed and some movement was present in the entire arm. When sitting quietly this tremor did not affect the leg, but the leg became somewhat affected during walking. Putting her hand on a flat surface, as on the table, the tremor ceased temporarily. The patient said also that the hand did not shake when she was lying in bed, awake or asleep. The movement was increased by a

* Read May 4, 1910.

† The symptoms here described are a summary of the results obtained on several examinations made during the week or ten days after she first came under observation.

volitional effort to hold up the arm away from the bed. Studying the movement more closely, it was seen to be slightly jerky, with a partial extending and flexing of the hand at the wrist. The head and face were not affected by the movement, but she had what she described as a sense of "drawing" in the right side of the face.

Some loss of power was present in the right upper extremity, and to a less degree in the lower extremity of the same side, as indicated by her ability to perform active movements and to resist passive movements. She had much difficulty in using the hand and fingers in performing finer movements, such as buttoning and unbuttoning her clothing. She showed weakness and awkwardness in the right arm in attempting the finger to nose test.

She was tested in various ways for powers of orientation and muscular sense. In having her blindfolded and attempting to move each arm through the same space, she did this awkwardly with the right arm and hand, although the space covered did not differ much on the two sides. Testing her by holding the fingers laterally and moving the parts on their joints, she was unable to repeat these movements or to reproduce the position properly with the unaffected hand. Besides testing with finger movements, sense of position was also studied by movements of the larger joints, as the wrist and elbow, with the same result, that is, the examination indicated the loss of the sense of position and passive movement.

Efforts were made to have her mark out on a ruler distances like an inch, or several inches, or parts of an inch, with the separated finger and thumb of each hand. She did this with fair precision with the unaffected hand, but in attempting to carry out the test with the right finger and thumb she exhibited much awkwardness. She marked the distance on the ruler nearly correctly, but her finger hovered and shook in the attempt. She had well-marked astereognosis in the right hand, not being able to recognize various objects with the fingers and hand of this side, these objects being promptly recognized by the left hand.

Several examinations were made to test her various forms of sensibility. The senses of pain and touch were not impaired. Spot localization was preserved. She did not always touch with precision the spot touched by the investigator. This was apparently due to uncertainty in the use of the unaffected hand rather than to real impairment of the sense of location. The sense of pressure was preserved.

One of the most interesting results obtained in this case was with regard to tactile discrimination. Testing with the compass points, the patient could not recognize them at twice the distance on the right hand, back and front, that she could on the left hand—indeed, she could scarcely recognize them as distinct points at any separation.

These tests were repeated with the same result at different examinations. The conditions as to impairment of power, sense of position and sensation were the same in the lower as in the upper extremity, but were not so marked.

Ptosis was not present and the ocular movements were not impaired. The right pupil, however, was somewhat larger than the left.

The following report by Dr. S. D. Risley, to whom the case was referred for ophthalmic examination, is of interest.

"A careful study of Mrs. C.'s ocular condition shows normal sharpness of central vision through correcting glasses. The fields of vision are also normal and the ophthalmoscope reveals only too full retinal veins, suggesting a possible increased blood pressure, but probably not too high for her age. The only thing I can discover about the eyes which may throw any light upon her intracranial disease is the fact that while both pupils react promptly, the right is larger than the left, the relative difference in size being greater in the diffused light of the consulting room than when facing a strong light with both eyes. This suggests an impairment of the light sense in the right eye, probably due to some disturbance, a stimulus possibly within the reflex arc or impaired perception at the oculomotor nucleus or higher up."

The urinary analysis showed an acid reaction, specific gravity at 15° C., 1023. A trace of albumin was present, but sugar was not found. The microscopic examination of the urine showed a few uric acid crystals and a few oxalate crystals; leukocytes present; squamous epithelial and numerous deep tissue cells; a few hyaline casts and various cylindroids.

This case is of much interest as the representative of a comparatively rare symptom complex. In the main it is that which is sometimes spoken of as the Benedikt syndrome. The lesion in this case was probably one involving the peduncle and the nucleus ruber. In lesions of the peduncle some form of oculomotor paralysis is present on the side of the lesion, with hemiplegia or hemiparesis on the opposite side. In connection with this, in the so-called Benedikt syndrome, a tremor which has sometimes been spoken of as like that of paralysis agitans may occur on the paretic or paralyzed side. The oculomotor trouble may be partial or total, or may even involve both sides, according to the extent of the lesion. In this case, if the dilated pupil is to be regarded as a form of oculomotor paresis, this involvement was very partial. According to some the tremor is rather of the intention type than of the type of paralysis agitans. If the lesion involves more of the tegmental region, hemiataxia may occur with, as in our case, loss of the sense of position. Leube[1] cites a case of hemiataxia, the oculomotor involvement consisting only of a constant ptosis and dilatation, the dilatation occurring later, but no other involvement of ocular muscles. Slight athetosis was present in both feet, but no distinct tremor or ataxia. Some sensory changes were also present on the paralyzed side in this case. Necropsy showed numerous small hemorrhagic areas in the peduncle and tegmentum.

Southard, in Vol. VII of Osler's *Modern Medicine* (Chapter XV, on Encephalitis and Brain Abscess), cites a case of Combe's illustrating a peculiar combination of symptoms in encephalitis with multiple foci: "That form of alternate paralysis known as Weber's syndrome," says Southard, "viz., right hemiparesis and left ophthalmoplegia, appeared in a boy aged two years. Weir Mitchell's[2] posthemiplegic choreiform movements followed on the right side, but associated with spasmodic movements in the field of the left facial and abducens nerves, amounting to an alternating hemichorea. Later there was weakness of the right oculomotor nerve. Such hemiparesis with crossed paralysis of the oculomotor nerve, with tremors of the paralyzed parts, corresponds with Benedikt's syndrome (due to a lesion of the cerebral peduncle), but the involvement of the left facial and abducens nerves, together with the right-sided hemiparesis, amounts to the Millard-Gubler syndrome. The patient, therefore, suffered from a combination of Benedikt's syndrome and the Millard-Gubler syndrome, due to a lesion of the pons near the left pyramidal tract (this new syndrome Combe proposes to call the inferior Benedikt syndrome), but in addition showed a complication by incomplete ophthalmoplegia, due to a separate lesion situated in the oculomotor nuclei."

This case was unlike the one here presented, in that it showed no sensory involvement and was located too far spinalward. It is, however, in some respects similar, as in the presence of the tremor on the hemiparetic side.

Bonhoeffer,[3] after referring to some cases of others and to two cases of his own, one with necropsy, speaks of the theories which have been advanced in the explanation of the hemiplegic or post-hemiplegic disorders of movement.

Kahler and Pick[4] believe that this movement was due to irritation of the pyramidal tracts.

Anton, cited by Bonhoeffer, published a case of bilateral choreoid movements, in which the necropsy revealed a bilateral lesion situated in the putamen of the lenticula. The same author, Anton, mentioned another case in which the lesion was in the thalamus.

Meynert and Nothnagel made similar observations earlier.

Anton believes in the existence of an antagonism between the lenticula and the thalamus. The lenticula, or certain parts of it, exercises an inhibitory influence over automatic impulses. Therefore, a destructive lesion of the lenticula interfering with the inhibitory influence would permit movements which would otherwise be controlled. The thalamus, on the other hand, exerts an accelerating influence over automatic impulses. The result, naturally, according as the lesion was irritative or destructive, would be the production or the suppression of the movement. According to this theory an equilibrium is established as regards coördinate movements between the lenticula and the thalamus. Anton calls attention to the observations of Freud, that the movements of infants and youthful children are of a choreoid nature, and also to the fact that in the diplegias occurring in childhood, such athetoid movements are of frequent occurrence.

Bonhoeffer calls attention to the well-known facts that in the early stage of hemiplegia choreoid movements are absent, and first present themselves with the return of some voluntary power, these increasing in proportion to the return of this power and the efforts at voluntary movement.

Bonhoeffer refers to an apoplectic case in which choreiform movements were present with marked hypotonia. This hypotonia was almost to the extent of a complete flaccidity. In this case, as in others, while impairment of power was present, it was not of the character which is seen in lesions of the pyramidal tracts.

Pineles, von Monakow, and Bernard Sachs also call attention to hypotonic flaccidity in cases of hemiplegic and post-hemiplegic chorea, so called. In ten of eighteen cases of Freud's with choreiform movements, contractures were absent. These facts, according to Bonhoeffer, contradict the theory that such movements are due to irritation of the pyramidal tracts. They are rather, according to him, due to interference with centripetal pathways. According to Bonhoeffer we have three positive reasons for the theory that choreoid-athetoid movements are due to lesions of the tract passing centripetally from the cerebellum to the motor cortex: (1) The constant occurrence in cases with these movements of a lesion of the superior cerebellar peduncle or its continuation into the subcortical ganglia. (2) The hypotonia of the musculature in such cases. (3) The disturbance of the voluntary movement occurring in chorea in addition to the involuntary movements present.

Bonhoeffer refers to the fact that Gowers had spoken of the occurrence of the athetoid movements in cases of cerebellar lesion. Von Monakow believed that impulses from the subcortical lesion passed to the motor cortex and there acted upon the pyramidal system in such a way as to cause the irregular (choreoid-athetoid) movements. Bonhoeffer agrees with von Monakow in so far that he holds that these choreiform movements are caused by interfering with the tracts which go from the tegmentum cortexward and under normal conditions serve to steady movements controlled by the pyramidal system. He believes, however, that while some tracts go from the tegmentum to the cortex, others pass into the subcortical ganglia (nucleus ruber and thalamus), and according as one or the other of these tracts is interfered with, will be the effect. If the cortical tract is cut off, the impulses will go by a short cut through the nucleus ruber, and in this way the incoördinate movements may be brought about. There is a flow of automatic movement in the ganglia, and according to the extent of the lesion will be the effect in motor symptoms.

He states that he had the opportunity of observing a case with accumulating symptoms and also impairment of power and movement gradually growing, in which, in the necropsy, he discovered a lesion about the size of a cherry at the decussation of the two superior cerebellar peduncles. Reasoning from this case and other data, he concludes that tracts pass from the cerebellum to the red nucleus, and other tracts from the cerebellum to the thalamus and to the lenticula, and that a lesion of any of these tracts might give rise to disorders of movement of a choreoid or athetoid character. It follows, therefore, that a lesion in the superior cerebellar peduncle, if situated at a certain part, would give rise to such manifestations. Bonhoeffer cites cases of Newton and Sander in support of his decision. In the former the lesions were in the neighborhood of the nucleus ruber, and in the latter in the region of the corpus dentatum in the cerebellum.

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THE SUDDEN ONSET OF PARALYSIS IN POTT'S DISEASE WITHOUT DEFORMITY OF THE VERTEBRÆ.

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Compression of the spinal cord causing paralysis may be due to various morbid processes, but here I shall refer only to compression following tuberculous caries of the vertebræ or Pott's disease. Although both the clinical and pathological manifestations of this disease have been carefully studied, there are still many disputed points in regard to the exact means by which interruption of the functions of the spinal cord are brought about, so for this reason, and on account of some very unusual features the following cases seemed worthy of careful study. I wish particularly to express my thanks to Dr. Spiller, Dr. Mills, and Dr. Frazier who have kindly furnished me with the material upon which this investigation is based.

Case I.—A colored male, aged thirty-eight years, was admitted to the Philadelphia Hospital in the service of Dr. C. K. Mills, November 7, 1904, complaining of paraplegia. His family history revealed nothing of importance. He denied syphilis. On August 7, 1904, he became very constipated, not having had a bowel movement for fourteen days; at the same time he had considerable abdominal pain. After evacuation of the bowels by means of purgatives, the pain subsided but still lingered. About one month later his right leg suddenly gave way while walking, associated with cramp-like pain. The pain persisted two days, and then the right leg became totally paralyzed. One week later the left leg was similarly affected but without pain. Again he became very constipated and had some difficulty in urinating. Since that time the patient has been confined to bed, and he says that he has not been able to feel anything in either lower extremity. Occasionally he had girdle sensation.

The following notes were made on the day of admission: The patient is fairly well developed. He can wrinkle his forehead evenly on both sides. The nasolabial folds are of equal prominence, he can show teeth, whistle, and draw mouth back on either side. The masseter and temporal muscles contract promptly. There seems to be some wasting of the face but it is symmetrical. Both pupils are round and equal and react promptly to light and accommodation. Sensation for touch and pain is normal over the face and neck. The extra-ocular muscles are normal. Motion and resistance to passive movements and grasp of hands is good on both sides. There is very slight ataxia of both upper limbs as shown by the finger to nose test. Sense of position is normal on both sides and sensation for touch, pain, heat, and cold is normal in the upper limbs. The lungs are hyper-resonant anteriorly and harsh breath sounds are heard over the upper portions. No rales can be elicited. The heart is normal. The abdomen is somewhat distended but not tympanitic. Palpation elicits no pain or tenderness. The abdominal organs seem normal. Sensation for touch and pain is normal over chest and abdomen; sensation for heat and cold is normal down to a point two inches below the umbilicus but below this level thermal sense is lost. There is marked atrophy of both lower limbs. Motion and resistance to passive movements is nil in both limbs. Patient is unable to move even toes. There is double foot drop. Impossible to test for ataxia on account of paralysis. Both limbs are slightly spastic, the left more so. At intervals there are involuntary contractions of the flexor and adductor muscles of each thigh. The right knee jerk is exaggerated, the left seems normal or slightly decreased. Ankle clonus is present on both sides, but it is much more persistent on the right. Babinski reflex is distinct on both sides. Sensation to touch is normal, on the left limb except for space extending downward six inches from the middle of the leg from that point downward it seems to be diminished, on the right side it is present, but the man is unable to locate touch on leg. Sensation to pain is present on the right limb but is diminished and he is unable to locate pin pricks. Sensation to pain on the left side is about the same as on the right. Sensation to heat and cold is absolutely lost over the whole of both lower extremities. Achilles jerk is absent on the right, but present on the left.

Two days later a slight angular deformity was discovered corresponding to the ninth thoracic vertebra. On November 22, all forms of sensation including touch, pain, and temperature were lost over both lower extremities and extended upward as far as the level of the umbilicus. A large bed sore had developed over the sacral region. The patient died November 30. The autopsy was performed the next morning.

Anatomical Diagnosis. Bilateral pleural adhesions, obliterative pericarditis, chronic fibrous tuberculosis of the lungs, malposition of the left kidney, gastrectasis, and enteroptosis, miliary tuberculosis of liver, dilatation and ulceration of œsophagus, subphrenic abscess, caries of lower thoracic vertebræ, tuberculous external pachymeningitis, internal hydrocephalus.

Microscopic examination of the lower thoracic segments where the pressure was greatest showed that the outline of the cord was not altered. The pia was slightly thickened on the anterior aspect, the adventitia of the pial vessels was less compact than normal and was infiltrated with small round cells. Here and there the pia was separated from the cord, the spaces between containing a few round cells and red blood corpuscles. In the white matter the neuroglia cells were swollen and increased in number; and the glia septa were thickened, standing out prominently. Many granular cells and a few round cells were seen diffusely scattered. The white matter presented the characteristic vesicular appearance. The medullary sheaths were distended, many axis cylinders had entirely disappeared and others were swollen or atrophied. The bloodvessels were thickened and there was moderate perivascular round-cell infiltration. Although the white substance was severely diseased the area of the lateral pyramidal tracts was perhaps more affected than the other parts. The central portions of the gray matter were entirely destroyed, including all of the gray commissures and part of the anterior horns, leaving a large irregular cavity with ragged edges containing debris, few small round cells, red blood corpuscles and compound granular cells. In the remaining portions of the gray matter there were scattered round cells and an occasional granular cell. Here the bloodvessels were also thickened, and there was moderate perivascular infiltration. The necrotic cavity did not extend beyond the limits of the eighth dorsal segment but the central canal in the adjoining sections above and below was distended and the ependyma cells were partially destroyed.

In the upper dorsal, cervical, and lower lumbar segments there was some thickening of the bloodvessels within the gray matter and slight perivascular infiltration. This was more marked in the branches of the anterior spinal artery near the base of the anterior horns. With the Weigert method there was degeneration of Goll's column extending upward into the cervical region. In the lower lumbar segments no distinct degenerations were found. At the level of compression many ganglion cells within the gray matter had been destroyed but a few remained which appear swollen, and with the Weigert stain the nuclei were indistinct. The Nissl stain could not be used. The bloodvessels of the pia about the pons and medulla seemed somewhat thicker than normal and some showed slight perivascular infiltration. The cells of the choroid plexus about the fourth ventricle were very much swollen and the bloodvessels were thickened. Sections of the cerebral cortex presented some rather doubtful round cell infiltration of the pia.

Case II.—J. D., was admitted July 16, 1904, to the service of Dr. Spiller, complaining of paralysis of both lower extremities. Three years previously his left leg had been amputated six inches below the knee as a result of injury. He denied syphilis. His family history was negative. July 2, 1904, he was compelled to go to bed on account of severe dull aching pain just below the angle of the scapula. Two days later there was much numbness and tingling just below the painful areas and on that day he suddenly lost power in both lower extremities. During this time he had had retention of urine and was constipated.

The following notes were dictated two days after his admission: The patient is fairly well nourished. Pupils are round and equal and react promptly to light and accommodation. The extraocular muscles are normal. Facial muscles contract equally and promptly; movements of upper extremities are equal and normal. The epigastric and abdominal reflexes are absent on both sides. Cremasteric reflex is absent on the left and diminished on the right. The knee jerks and Achilles jerks are absent on both sides. There is no ankle clonus on either side. Sensation for pain and touch is lost over both legs and abdomen up to a point two and one-half inches below the right nipple, and about one inch lower down on the left side. Adjoining this line above is a zone of hyperesthesia about two inches wide, which narrows to about one inch in width near the axilla of either side. Testicular pain is absent.

The following additional notes were dictated by Dr. Spiller some days later: Sensation for touch, pain, and temperature is similar to the above. Cremasteric reflex is present on both sides but diminished. Passive movement of the stump of left leg on thigh causes involuntary jerking movement of this limb of which the patient is not conscious without seeing it, but he is conscious of a jarring sensation in his back. These involuntary movements are not distinct in the right lower limb. He has no knowledge of the sense of position of his legs. The knee jerks are both absent, the plantar reflex is present on the left side, but sluggish on the right. There is an area of tenderness of the spinous processes about the level of the spines of the scapula, but no deformity.

The patient died July 28, 1904.

Macroscopic examination of the spinal cord and its membranes showed an external pachymeningitis extending from the lower cervical region to the upper lumbar. There was a considerable amount of necrotic cheesy material which by microscopic examination revealed the characteristic picture of tuberculous disease. Microscopic examination of sections taken from the upper thoracic portion of the spinal cord showed no diminution in its volume.

When stained by the hemalum and fuchsin method there was slight cellular infiltration of the pia, and the pial vessels presented slight infiltration of the adventitia. In the white substance there were numerous vacuoles, mononuclear cellular infiltration, thickening of the bloodvessels and slight perivascular infiltration. The neuroglia cells were increased and here and there were a few granular cells. The anterior portions of the posterior columns were partially destroyed. The anterior longitudinal septum was infiltrated with round cells and appeared more prominent than normal. In the gray substance there was moderate diffuse cellular infiltration of the mononuclear type and red blood cells as well as compound granular cells were found. The blood vessels which were cut in cross section appeared thickened, and the perivascular and periganglian spaces were enlarged. The most marked inflammatory changes were found about the gray commissures and central portions of the anterior horns. In some sections necrosis has taken place in the area supplied by the commissural branch of the anterior spinal artery. With the Weigert method the posterior and lateral columns suffered most, and the anterior columns least. Axis cylinders had disappeared and many remaining were swollen, while here and there a few normal fibers were seen. Many of the ganglion cells of the anterior horns had disappeared, others stained poorly, and their nuclei were indistinct. In the lower thoracic lumbar and cervical segments the process was similar in the gray matter, but much less marked, while the white substance was little affected. Distinct tract degeneration was absent except for a few degenerated fibers of Goll's column. Sections of the medulla in its lower part showed very minute capillary hemorrhages chiefly in the gray matter. The brain was not examined.

Case III.—C. M., a white female, aged fifty years, was admitted to the Philadelphia Hospital in the service of Dr. Spiller complaining of weakness of the lower limbs. Syphilis was denied. Three months previous to admission patient first developed some pain about the left shoulder which resisted all treatment. About two months later both legs began to grow weak, this gradually progressed until one week before admission when she was unable to walk or stand without assistance.

Physical Examination: The patient is much emaciated. The pupils are equal and react normally to light and accommodation. Movements of muscles of the face, tongue, larynx, and pharynx are normal. The power of the upper extremities is equal and normal. Triceps and biceps jerks are equal and normal. Both lower extremities are completely paralyzed and there is extreme contracture at the knees and hips. From time to time there are violent jerking movements at the knees. Patellar tendon reflex is normal on the left, but slightly decreased on the right. Achilles reflexes are equal and normal. Clonus is absent. There is distinct

Babinski signs on both sides. Sensation for pin prick and for touch is lost over both lower extremities and abdomen as far as the fifth interspace, at this point pain and tactile sensations are impaired but not lost. Sensation for heat and cold was not tested. The vertebral column although prominent on account of emaciation showed no deformity.

Macroscopic examination showed tuberculous caries of the second thoracic vertebra and an external tuberculous pachymeningitis beginning one quarter of an inch above the exit of the second thoracic root and extending one quarter of an inch below the exit of the third thoracic root, therefore involving the whole of the intradural portion of the third thoracic root from its exit from the cord to its exit through the dura. Microscopic examination of section taken from the third thoracic segment showed no change in contour. When stained by the hemalum and fuchsin method the pia seemed normal, but there was a slight mononuclear cellular infiltration of the adventitia of some of the pial vessels. In the white matter the walls of the bloodvessels were thickened and there was a very slight perivascular infiltration of mononuclear cells and an increase of the glia cells. There were no distinct areas of softening.

In the gray matter there were many mononuclear cells diffusely scattered. By the Nissl stain the ganglion cells were few in number and those remaining were much degenerated. With the Weigert method the medullary sheaths were swollen and many axis cylinders had disappeared. By the Marchi method nearly all the fibers of the white matter were degenerated, but a few scattered normal fibers still remained on the lateral columns near the periphery. Above and below the site of compression distinct tract degeneration was absent, though a few fibers were found staining very darkly in the pyramidal tracts and in the posterior columns by the Marchi stain.

In cases of tuberculous caries infection is generally carried to one of the vertebræ from some other focus within the body such as the lung, and a chronic osteomyelitis or periostitis ensues. This may go on to necrotic softening, and then by purely mechanical means the adjacent vertebræ above and below become approximated producing the well-known deformity of Pott's disease. In some instances this deformity may be sufficient to cause direct pressure upon the cord, but as Kraske has shown, it is uncommon; he having found it in only 6 of his 58 cases. Occasionally a subperiosteal abscess may exert pressure; Oppenheim and others have even seen cases in which spicules of necrotic bone encroached upon the cord. In the great majority of cases however, infection extends to the external lateral and posterior surface of the dura setting up a chronic inflammation which produces great thickening with epidural exudates and fungoid proliferations. Fickler in 20 cases found compression caused by dislocation of a vertebra in 9 per cent.; by abscess formation, in 17 per cent.; while pachymeningitis externa was responsible for 73 per cent. Infection of the dura nearly always arises by direct extension from a focus in one of the vertebræ, yet Schlesinger, Henneberg, and Rossi, have each reported a very uncommon condition in which there was tuberculous pachymeningitis without caries of the vertebræ. A similar case has recently been under the care of Dr. Spiller, and Dr. Frazier, the notes of which are as follows:

Case IV.—H. B., a male, white, was admitted to the University Hospital, April 10, 1909, in the service of Dr. Spiller.

The patient's father, grandfather, one aunt, and one sister died of tuberculosis. Six years ago the patient had scarlet fever which was followed by paralysis of all four extremities lasting two months and then he entirely recovered. Seven months previous to admission he first developed pain in the region of the sixth dorsal vertebra. Pain was present on movement and on jarring of the spinal column, and continued in spite of treatment. About five weeks ago he noticed his legs were growing weak and that he staggered when walking. Occasionally he had cramp-like pains in the calf muscles of both legs. This condition increased gradually until two weeks ago when he was not able to walk at all. Pain still continued in the lower legs. Patient cannot control bowels and can retain urine only a short time, and is troubled with distention of the abdomen. He states that for the last four weeks he could not feel distinctly below the hips.

The following notes were dictated by Dr. Spiller: Patient is completely paralyzed in both extremities, slight movement of drawing upward of the left lower leg seems to be from muscles of the trunk. Limbs are not wasted. Patella and Achilles reflexes are about normal on each side. No ankle clonus on either side. Dr. Willard, Jr., noticed bilateral ankle clonus three days ago, before patient entered hospital. Tactile sensation seems to be preserved in all parts of the body except possibly over the front and outer side of the thighs. Here he sometimes answers incorrectly. Pain, heat, and cold sensations are lost or greatly impaired over both lower limbs and trunk anteriorly and posteriorly to a line three inches below the nipple. Area of disturbed sensation shades off gradually into an area of normal sensation. Babinski sign is typical on each side. Testing him with pin prick, or heat, and cold, causes at times either lower limb to be forcibly drawn upward. The upper thoracic vertebræ are distinctly arched, but there is no sharp kyphosis, only gradual arching. Von Pirquet tuberculin test was positive, twenty-four hours. Urine was negative. Blood count was: hemoglobin, 48 per cent.; red blood corpuscles, 4,880,000; leukocytes, 11,900.

Additional notes dictated by Dr. Spiller, May 19, 1909: Both legs are completely paralyzed. There is considerable desquamation of the skin of the feet and legs below the knee. There is involuntary drawing upward of lower limbs at hips and knees. Has imperfect control over bladder, cannot hold urine; no control over rectum. Only pain he has now is at lower costal margin, and there is no atrophy of any part of the body or even of the lower limbs. Lower limbs are very spastic. Knee jerks are exaggerated on both sides. Patellar clonus present on both sides. Achilles jerks are exaggerated on both sides but clonus is not obtained on either side on account of spasticity. Any irritation of the limbs or trunk causes limbs to be drawn forcibly upward. Touch, pain, heat, and cold sensations are lost in the lower limbs and trunk anteriorly and posteriorly to a line drawn around the body from the eleventh and twelfth thoracic vertebræ to the sixth interspace on the left side in the nipple line, and seventh interspace on the right side in the nipple line. This line is very sharp for touch but not so sharp for pain and temperature sensations. There is no involvement of the upper limbs. Prominence of vertebræ is in the midthoracic region but no actual displacement.

On June 24, Dr. Frazier removed the fourth, fifth, and sixth dorsal spinous processes. During the operation the following notes were dictated. Coming out between the transverse processes of the fifth and sixth dorsal vertebræ was found a mass of tissue about the size of a marble presenting the appearance of granulation tissue. Upon exposure of the cord it was found free from involvement, the tumor tissue seemed to come out from either right side of the body of the vertebra or from the transverse process of the fifth dorsal. All tumor tissue that could be was removed with scissors and curette. There was evidently compression of the cord at the site of injury which was relieved by removal of the laminæ. Tumor did not make pressure over the dorsal but over the lateral aspect of the cord. Recovery after operation was good.

The pathological report was as follows: The specimen consists of laminæ and small portion of spinal meninges with nerve cord about three inches long. Microscopically, soft tissue removed from laminæ shows simple areolar tissue, the fibrous elements of which are somewhat thickened and in places infiltrated with small round cells, also a few degenerated muscle fibers are seen and many typical tubercles were found with epithelioid cells and giant cells.

On October 11 examination of the patient showed there was no return of power in the lower extremities. Sensation to pain and touch is lost below the fifth interspace on the right side anteriorly and the sixth space anteriorly on the left side. Patient complains of sharp pain in the region of the right nipple on attempting to raise head from bed.

In attempting to explain the intraspinal lesions of Pott's disease it must be borne in mind that there are two cardinal factors each of which originate outside the dura, namely, pure mechanical compression and infection. Charcot and Michaud believed that degeneration of the spinal cord was caused by infection extending into the substance of the cord setting up a secondary myelitis; on the other hand, Kahler was able to show by compressing the spinal cords of animals with non-toxic substances that lesions similar to those following caries of the vertebræ resulted. Later Leyden, recognizing the possibilities of infection, declared that compression in itself was an important factor. Strümpell strongly opposed the myelitic theory explaining the alterations in the cord as the result of œdema stoppage of the lymph stream, and anemia, secondary to pure compression; Schmaus was inclined to accept Strümpell's view but modified it somewhat by adding that there was a collateral inflammatory œdema which could be attributed to toxic influences; more recently, however, in an article upon myelitis the same author has expressed the opinion that constant prolonged pressure upon the cord may lead not to stasis œdema but to true secondary inflammation, that is, myelitis.

The first three cases which I have described presented swelling and destruction of axis cylinders, proliferation of the glia, round-cell infiltration, sclerosis of the bloodvessels, perivascular infiltration, necrotic softening of the central gray matter, and compound granular cells at the level of greatest compression of the spinal cord. In Case I there was an internal hydrocephalus affecting only the lateral ventricles; the formina, however, were not occluded, but the cells of the choroid plexus were decidedly diseased; it is conceivable, therefore, that as a result of perverted function of diseased cells there may have been an increase of secretion sufficient to cause this distention. But it is also possible that hydrocephalus may have been the result of compression of the cord, and that if an ophthalmological examination had been made choked disk would have been found. Choked disk has not been seen frequently in Pott's disease, yet it has been observed in spinal tumor by Bailey. At other levels of the spinal cord where compression seemed to play no part there were definite though very moderate signs of vascular disease, consequently it might seem as if there was some other element responsible as well as compression.

Spiller in an article on Pott's disease refers to a form of simple meningomyelitis without giant cells or miliary tubercles, occurring particularly when Muller's fluid had been used for preservation, he also quotes Oppenheim who had called attention to a similar type of myelitis in persons dying of generalized tuberculosis. In my cases there were no specific signs of tuberculosis found within the dura, yet the diagnosis of Pott's disease is evident, and it does not seem probable that the existence of myelitis would be disputed.

The symptoms of cord involvement in Pott's disease vary considerably in their mode of onset, but after having once begun they frequently progress rapidly. Usually the onset is chronic, the signs of paralysis requiring some months to reach their height. Occasionally paraplegia develops in the course of two or three weeks. Very rarely, however, there is an instantaneous onset, and, moreover, in such cases there is usually a history of trauma, or some undue strain upon the diseased vertebral column immediately preceding the initial paresis or paralysis, which causes sudden displacement of one of the vertebræ and thus brings about rapid compression of the spinal cord. Gowers describes a child with angular deformity of the spine, who, while walking across the room, suddenly fell to the floor and after being lifted up was paralyzed in both legs. In Case IV not only was deformity absent, but, as far as could be determined at operation, the vertebræ were entirely normal. Deformity was entirely lacking in the other three cases at the time of the onset of paraplegia, yet in Case I and Case II the development of paralysis could not have been more rapid. In the first case, motor and sensory paralysis of one lower extremity was said to have begun instantaneously, the patient's right leg suddenly giving way under him while walking, and one week later the left leg was similarly affected. The second patient developed paralysis of both lower extremities in an equally rapid manner. Truly an apoplectiform onset in the strictest sense of the term.

After searching the literature upon this subject I have been able to find only two cases described in which paralysis developed instantaneously without deformity of the vertebræ. One case with necropsy reported by Raymond seventeen years ago seems to have been entirely overlooked, another without necropsy was described by Martineck during the past year. Raymond's case is as follows: A young girl developed complete motor paralysis of both upper extremities and incomplete motor paralysis of the lower limbs within a few hours. Laminectomy was performed and the pia was found to be thickened and the cord much congested, but without evidence of compression. The patient died on the same day and at the autopsy tuberculous caries of the third cervical vertebra and abscess formation was found. Microscopic examination showed inflammation and hemorrhage in the gray matter, sclerosis of the bloodvessels and cellular infiltration of the pia, while the white matter was much less involved, yet there were no specific lesions of tuberculosis within the dura.

Martineck's case was that of an adult male, who had been complaining for some months of severe pain. The patient went to bed feeling generally weak, the next morning there was paresis of both legs, by the afternoon there was paralysis of both lower extremities. When the patient was seen at the hospital the following day there was almost complete motor paralysis and involvement of the bladder, still the vertebral column showed no abnormality. There was neither tenderness nor pain on movement. It was not until eight days after paralysis had developed that an angular deformity was discovered extending from the eighth to the eleventh dorsal vertebræ. *X*-ray examination showed abnormality in the position of the ninth dorsal vertebra.

When the term myelitis is used in its strictest sense to mean inflammation of the spinal cord, Russel believes that it is a rare disease, and attributes much of which we speak of as myelitis to softening consequent upon vascular occlusion, in which inflammation plays no part, unless it is some inflammatory process in connection with the walls of the bloodvessels which tends to thrombosis and later softening. Douglas Singer also refers the majority of cases of myelitis to primary thrombosis.

It is well known but perhaps not thoroughly appreciated, that in cases of syphilis occlusion of some of the smaller spinal vessels produce sudden weakness and paralysis; in fact, sudden onset of paralysis is common in syphilitics. Spiller, in an article upon syphilitic acute anterior poliomyelitis due to thrombosis, emphasizes the importance of considering the rapid development of paralysis and says "spinal thrombosis is not a rare condition and is probably the cause of most of the apoplectiform palsies that occur in myelitis." In my case, syphilis, dislocation of vertebræ and trauma can all be excluded with certainty, yet a traumatic cause, if this expression is permissible, was present, that is to say compression. As has been already pointed out, compression of the spinal cord even when slight, can in itself cause œdema and inflammation with thickening of the bloodvessels within the cord, in other words, a fertile soil for thrombosis. Epidural inflammation and fungoid proliferations increase very gradually up to a certain point without evident disturbance in the functions of the cord unless it be pain; but when this point has been reached a very minute sudden increase may be sufficient to give signs of rapidly developing weakness or palsies, on the other hand, it is possible that it is just at this stage of the disease that thrombosis develops. Whether or not in my cases the initial palsies were caused by sudden increase of pressure with thrombosis, or to increased pressure without thrombosis, or to the changes which were found within the cord, is of course, problematical.

Raymond was inclined to look upon his case as one of hematomyelia, also Huber and Vorkarstner in discussing Martineck's case suggested the possibility of there having been a hemorrhage within the gray matter of the cord in addition to compression. Spontaneous hemorrhage in the spinal cord is exceedingly rare. Minor who has studied this question minutely, says that among all the cases which he has observed (other than Pott's disease) hemorrhage occurred only twice without preceding trauma. In an anatomical sense it is always difficult to make a sharp distinction between primary hemorrhage with subsequent myelitis from primary hemorrhagic myelitis and in many instances absolutely impossible.

In Cases I and II it was impossible to prove conclusively that thrombosis had taken place, nevertheless on account of marked thickening of the bloodvessels throughout the cross section of the cord, together with extensive central softening and similarity of the onset of paralysis to cases of primary myelitis in which thrombosis has been proved, it seems more than probable that thrombosis may have occurred. At all events, instantaneous paralysis in Cases I and II and the more slowly developing paraplegia in Cases III and IV was the clinical expression of an intense myelitis and was beyond all doubt the direct result of compression of the spinal cord by means of tuberculous external pachymeningitis.

In determining the upper limits of a focal lesion causing compression of the cord, particularly when situated in the dorsal region, there is always more or less uncertainty. The most important single sign is the character and extent of sensory loss of function. Certain authors rely upon the level of hyperesthesia so frequently found just above the level of the total loss of sensation. In an article on spinal tumors Bailey maintains that the upper limits of incomplete loss of sensation is probably the most accurate guide in determining the exact level of the lesion. In two of my cases the lesions were too diffuse to draw accurate conclusions, but in Cases III and IV the limits of hypesthesia of the skin indicated quite accurately the segment of the cord which was involved.

The diagnosis of Pott's disease when deformity of the spine is present is generally easy, but, as Moussaud has pointed out, without this characteristic sign an accurate diagnosis may be exceedingly difficult or impossible. Fortunately, cases of this kind are uncommon, yet unmistakable evidence of caries is by no means always present. Nevertheless by a careful consideration of the clinical history, bearing in mind the similarity of the onset of paralysis in certain cases of Pott's disease with that of other form of myelitis particularly in syphilitics, and by use of *x*-ray examinations, tuberculin test, the Wassermann and Noguchi reactions, and lumbar puncture, in a certain number of cases at least, an accurate conclusion can be reached. If in a given case it can be proven that the bones of the vertebral column are not involved and the initial paralysis did not begin suddenly operation should be seriously considered. In such cases it is probable that the symptoms are caused by slowly increasing pressure upon the cord by a dry inflammatory exudate with a minimum amount of pus, or pus may be entirely absent as in Case III; therefore, if operation is done early and pressure removed recovery might be possible. Recovery does not seem to occur without operation. On the other hand, an apoplectiform onset of paralysis would indicate serious intraspinal lesions and operative interference might be of no advantage, and yet it is probable that in these cases also, compression is the primary etiological factor; if, therefore, it can be removed by operation, the injured cord will have better opportunity to recover its normal function, although in Case IV operation was of little benefit.

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Diagnosis and Medical Treatment of Poliomyelitis.*

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The recent study of poliomyelitis has added much to our knowledge of the disease. We have learned among other things that subjective sensory symptoms, as pain, are common, that the disease at least clinically may be multiple neuritis even though the pathological findings have not as yet confirmed this diagnosis, that adults at least in the epidemic form often are attacked, that abortive cases without any paralysis are common, and that complete recovery even when paralysis develops may occur. Unfortunately we do not yet know the cause of the disease, we know little about preventive means, and our curative remedies are conspicuous in their deficiency.

ONSET.

Fever is common. It is not usually high; lasts only a few days to a week, but may recur or may persist as long as two weeks. The intensity of the fever and of the other initial symptoms is not indicative of the course of the disease, as severe symptoms may usher in a mild case, and mild symptoms be the beginning of a severe case. Chill seldom occurs. General symptoms as fatigue, headache, vomiting, somnolence, pain of body, rigidity of neck and severe sweating are common. Unconsciousness and convulsions are rare. Pain is now recognized as a common symptom; it may be spontaneous or produced by slight pressure. Diarrhea is more common than constipation. Disturbances of the respiratory tract, as nasal catarrh, angina, mild or severe bronchitis, and occasionally bronchopneumonia, may be associated conditions. Sometimes there is swelling of the joints. By some, leukopenia has been observed in the early stage. The leukocytes are not increased in the fever stage, and in some cases they are diminished. Morse[1] says the leukopenia, which occurs in animals during the early part of the acute stage, is certainly not a constant symptom in man, and in many instances it is replaced by a hyperleukocytosis. The leukocyte count is of little or no assistance in the early diagnosis of acute poliomyelencephalitis. There is at present not sufficient evidence to show whether or not there is a relative or absolute lymphocytosis in the early stages. Retention of urine and feces may occur but seldom is persistent and is more common when the lower limbs are paralyzed.

The pressure in the cerebrospinal fluid may be increased, even some weeks after commencement of the disease, and the fluid may show an excess of albumin. Some observers have found a diminution of lymphocytes in the fluid, others an increase of lymphocytes and even of leukocytes.

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* This paper was read as part of a symposium on Poliomyelitis.

[1] John Lovett Morse: *Archives of Pediatrics*, March, 1911, p. 164.

John Lovett Morse, who has devoted much attention to this subject, says that during the acute stage of this disease the cerebrospinal fluid is clear and not infrequently under somewhat increased pressure; it often shows fibrin clot which may persist two or three weeks or longer; it always contains an excess of cells, and these are chiefly of the mononuclear type, most of them being lymphocytes, and these changes are present before the appearance of the paralysis.

Flexner has found an increase of cellular contents, chiefly lymphocytes, and an excess of protein in the cerebrospinal fluid, determined by the Noguchi butyric acid test, in the early stage of poliomyelitis. The fluid may also be opalescent. This condition may indicate that paralysis is likely to follow. In monkeys, the increase in lymphocytes and increased protein reaction are present in the spinal fluid only a few days, and it is probable that the same is true of man.

Lafora's[2] findings are similar. According to Lafora the cerebrospinal fluid is generally clear; in the early stages there is a moderate increase of pressure; the polymorphonuclears are at first increased but this increase disappears a few days after the acute onset of the disease and is substituted by a lymphocytosis with some plasma cells and sometimes a few mast cells.

The paralysis is the most characteristic sign of the disease. It is seldom complete from the beginning. In early infancy the initial weakness may readily be overlooked, as it is not at first detected in a child who has never walked or stood. If a careful observation of the case be made, weakness will usually be found to precede paralysis by one to several days. The patient becomes more easily fatigued, desires to rest after moderate exertion, but the complete paralysis may develop rapidly. Often several days or a week are required for its full development. It is seldom progressive over a period of two or more weeks, but in rare instances it has been observed to be progressive during several months (Neurath). Doubt regarding the correctness of the diagnosis always exists in a case of this kind. The paralyzed muscles are almost always flaccid, but may in rare instances be spastic. As the pathological process is a meningomyelitis attacking chiefly the gray matter of the cord, it produces flaccidity and loss of tendon reflexes, but in the cerebral types of the disease or where the cord is implicated chiefly above the lumbar region, the tendon reflexes of all the limbs in the former variety and of the lower limbs in the latter variety may be exaggerated. I have observed exaggeration of tendon reflexes in the lower limbs in at least three cases of the spinal form of the disease, and it has been observed by others. The paralysis may implicate any group of muscles in the limbs; there are, however, certain muscles which are especially susceptible to the paralysis. In the lower limbs the peroneal group and the quadriceps femoris muscle are likely to be paralyzed, and in the upper limbs the deltoid muscles and those of the upper arms are more commonly affected.

The paralysis soon reaches its height and is attended in certain muscles with loss of faradic irritability, which however is not a reliable sign of incurable paralysis. Certain muscles gradually recover, and in some instances the paralysis entirely disappears. In rare instances recurrence of symptoms is seen, and the interval between the attacks may be of weeks' or even months' duration, but usually one attack gives immunity.

Some of the causes to which poliomyelitis is attributed are difficult to accept. I have jective sensation was not affected. The calves were still tender to pressure. The left lower limb when she was examined by me, February 9, 1910, was much more paralyzed than the right. She still had aching of the entire left lower limb. The power of the upper limbs was nearly normal. This case was seen also by Dr. Mills.

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[2] Lafora: Report of the Poliomyelitis Committee of the Med. Asso. of the District of Columbia, Epidemic of 1910. *Washington Medical Annals*, Vol. x, No. 2, May, 1911.

Landry's paralysis: This type is well recognized; indeed Landry's paralysis is usually poliomyelitis or multiple neuritis, and as it is not very common, it follows that the poliomyelitis form is even less frequent. The paralysis begins in the lower limbs, and within a few hours or a few days extends to the upper limbs. It is always flaccid, with loss of tendon reflexes, and if it persist long enough, may be with change of electric irritability. I have known recovery to occur in a case of this type with intense paralysis of all four limbs.

The ataxic type: It is questionable whether this type should be recognized as distinct from the encephalic type. When the cerebellum or the tracts in the brain and cord connecting with it are implicated, ataxia may result, but other parts of the brain and cord are almost invariably involved in the lesions, and other symptoms, as paralysis of cranial nerves, are therefore likely to occur. We must recognize that the acute ataxia of the German writers may be produced by poliomyelitis, but if it occur it must be extremely rare and can cause little confusion in differential diagnosis. In the ataxic type the tendon reflexes may be exaggerated or lost, and atrophy may not develop, as the lesions are chiefly above the cord.

A herpetic type possibly may be established, although some maintain that herpes is not in any way related to poliomyelitis. Much the same lesions are found in the spinal ganglia as in the spinal cord. Garrow[6] observed an unusual number of cases of herpes zoster during August and September, 1910, when acute poliomyelitis was epidemic. Müller[7] acknowledges that the histological changes in the cord and spinal ganglia are much alike, and yet he has not noticed an increase of zoster during an epidemic of poliomyelitis, nor has he seen poliomyelitis associated with zoster.

Late atrophic type: when the acute stage is over, recurrence of paralysis is rare, but a progressive muscular paralysis may develop years after the apparent cessation of all symptoms. Pastine[8] is a recent writer on this subject. He has studied two cases, in one of which the atrophy began seventeen years after the attack of acute poliomyelitis, in the other seventy-five years later; the latter case was with necropsy, and the cause of the late atrophy was chronic anterior poliomyelitis. Cassirer has found as the cause of the late atrophy, progressive muscular dystrophy; Oppenheim has found disseminated myelitis and occupation atrophy. Two cases of this type have come under my observation, the more remarkable is the following:—

Nelson G., aged fifty-seven years, was admitted to the University Hospital under my care, March 31, 1905. He had previously been in my service at the Philadelphia Hospital. He stated that he had been well until he was three years of age. At that time his right leg became paralyzed and he was unable to use it for some time. Later he regained power to the extent that he could walk. The paralysis probably was caused by poliomyelitis.

In 1901 he had an abscess in the palm of the left hand, and the left hand, soon also the right, began to grow weak; his arms also became weak.

The right lower limb was much smaller than the left, especially below the knee, and the muscles of the calf were flabby. The right foot was in the position of talipes equinovarus, and all movement of it was lost; in the right toes only slight flexion was present. The patellar and Achilles reflexes were lost in the right side, exaggerated in the left side, and persistent ankle clonus was obtained on the left side. Sensations of touch and pain were normal in all and the patient left the hospital. Both these cases were seen by Dr. Mills.

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[6] Garrow: *British Medical Journal,* March 18, 1911, p. 621.

[7] E. Müller: Die Spinale Kinderlähmung, Berlin, 1910.

[8] Pastine: *Revue Neurologique,* 1910, p. 466.

It is not known whether these students had been together, but both were attacked about the same time and when poliomyelitis was prevalent. Hemiplegia in a young, healthy male, with healthy heart and kidneys, and without syphilis, is unusual. The rigidity of the neck, the tenderness of muscles and nerves, the leukocytosis, the low-grade papilledema are suggestive of the encephalic type of poliomyelitis.

A case illustrating the peculiar distribution of paralysis, being both spinal and bulbar, that poliomyelitis may assume was one seen by me in consultation with Dr. Tracy of Beverly, N. J., September 17, 1910. Such combination is not common.

A man, twenty-one years of age, about July 20, 1910, had an illness beginning with headache, nausea and vomiting, soreness in all his limbs and back, and temperature 101°, in four days reaching 103°. He had visited a child a week before he was taken sick, who, four days after the visit, developed the symptoms of poliomyelitis, this suggesting an incubation period of one week in the man. He did not notice weakness of the right lower limb until about August 3, when he got out of bed, but it is possible that the right lower limb was becoming weak while he was in bed. About August 3 he could not bite well; a few days before this date speech had become a little indistinct.

In my examination, September 17, 1910, I found that the right lower limb was distinctly weak, the right patellar reflex was feeble, and the left patellar reflex was exaggerated. Babinski's sign was not with upward turning of the toes. Speech was still slightly affected. The masseter muscles were very weak, and the lower jaw deviated a little to the left when the mouth was opened. In attempting to close the jaws the teeth did not touch. The facial nerve supply was not affected. Sensation of the face was normal. Slight nystagmus was present in extreme lateral movements of the eyeballs.

The paralysis was peculiar in the marked weakness of the right lower limb and of both masseter muscles, with complete integrity of the sensory fifth distribution, and exaggeration of the left patellar reflex.

The meningeal type is common, although it is questionable whether pain and tenderness to pressure are so frequent as in cases observed by Wickman. The pain in the head and back, stiffness of the neck, tenderness of the muscles on handling, Kernig's sign, etc., are very suggestive of meningitis, and meningitis is not an uncommon finding in association with poliomyelitis. The meningeal type is represented in the hemiplegic case described above.

The polyneuritic type is seen occasionally. In this, spontaneous pain and pain on pressure are pronounced; there may even be objective sensory disturbances in rare cases, and especially of the sensations of pain and temperature, from implication in the posterior horns of the fibers concerned with these sensations. While we recognize clinically the neuritic form, we have not pathological evidence justifying this recognition, as no changes in the nerves have been noted. I believe, however, that this failure of observation is not from failure of neuritic changes, but from inability to study the nerves in cases in which neuritic symptoms have been pronounced. It is possible that the meningitis may explain the pain.

One of the most striking cases of painful poliomyelitis is the following, sent to the University Hospital by Dr. J. W. McConnell. The patient was referred by Dr. E. Wills.

A woman, aged twenty-four, living near Pottstown, on July 27, 1910, perspired profusely during the night, then had sharp shooting pains all over the body, especially down the back and in the region of the kidneys. She had fever. She asserts that paralysis developed within five minutes. She walked from one room to another and sat down, and about five minutes later she could not stand, and from that time had not walked. She passed no urine the day following the development of the paralysis. Great pain persisted in all parts of the body, and insomnia was severe. The lower limbs were very tender, and when she was admitted to the hospital, January 27, 1910, she still had pain and the extremities were cold. The lower limbs at this time were very weak, flaccid and atrophied; the patellar reflexes were lost. Obsymptoms, but purely encephalic forms of the disease occur. There may be hemiplegia when the cerebrum is attacked, or bulbar palsy, or some form of polioencephalitis, superior or inferior, when the process is most severe at the base of the brain. More or less complete ophthalmoplegia, usually unilateral, has been observed, even isolated ptosis, also isolated facial palsy, usually unilateral. Almost any combination of cranial nerve palsy seems possible, and yet these encephalic types are rare and have not received the attention they deserve. When they occur independently of an epidemic they seldom are recognized as belonging to poliomyelitis. The facial nerve is more frequently affected than any other cranial nerve, and usually the palsy is unilateral; indeed, facial palsy may be the only paralysis from poliomyelitis. The encephalic form is not easily produced experimentally; even when the injection is intracranial the symptoms are spinal and cerebral paralysis has not been produced in the ape by most experimentors unless abscess occurred. Römer,[4] however, has recently stated that experimental poliomyelitis in apes may assume the form of Landry's paralysis, a bulbar, pontile or a pure cerebral form.

If we accept the dictum of Sydney Stephenson,[5] the encephalic form is more common than it is usually believed to be. He says the clinical material at his disposal is not especially large, yet he has collected twenty-eight cases of this type. He concludes that there is a particular form of paralytic strabismus in children which is due to poliencephalitis; it is most frequent in children under one year of age; it is comparatively seldom associated with other symptoms indicative of cerebral disorder; and zymotic diseases appear to be important factors in its causation. The paralysis may affect any of the extrinsic muscles of the eyeball, yet in three fourths of the cases the external rectus muscle is alone involved. The common form of encephalitic strabismus is likely to be confused with the ordinary form of concomitant convergent strabismus.

These conclusions seem to be deduced from clinical evidence, and it is questionable whether all cases of ocular palsy occurring during or following an infectious disease, such as measles, should be included under the term poliomyelitis, or even the encephalic type of this disease.

As an illustration of the cerebral form of poliomyelitis I may refer to a case that was in the students' ward at the University Hospital under the care of Dr. Edsall. I had the opportunity of examining this patient several times. Another student, twenty years of age, affected with poliomyelitis, had been admitted to the hospital, October 17, 1910, under Dr. Musser's care. His symptoms began soon after he had had severe foot-ball practice, and the case illustrates what I have observed in other cases, viz., that severe exercise sometimes has preceded the onset of poliomyelitis in adults. The cerebral case was as follows:—

A man, aged twenty years, admitted to the students' ward, October 19, 1910, rapidly became hemiplegic and unconscious. His cardiac action was normal. The kidneys were healthy. The neck was stiff. White blood corpuscles numbered 23,200. Lumbar puncture was dry. Three days after admission he had pain on motion in the back of the neck, distinct tenderness along the left brachial plexus, tenderness in the muscle masses and along the nerves on the right and left sides, especially in the right arm, shoulder and calf; pain in the back of the head, and distinct rigidity of the neck. On the fourth day after the onset the white corpuscles had diminished to 15,100. Dr. deSchweinitz found low-grade papilledema in the right eye, the nasal, upper and lower edges of the disk were hidden, and the veins were slightly larger than normal. The condition in the left eye was similar but less intense. Improvement occurred the notes of a case observed by me in 1900, in which a woman developed the symptoms of Landry's palsy shortly after she had employed a vaginal injection for the purpose of abortion. All four limbs were paralyzed and breathing was difficult. This woman made a complete recovery with the exception of a slight weakness in one small group of muscles.

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[4] Römer: *Monatsschrift für Psychiatrie und Neurologie*, July, 1911, p. 69.

[5] Stephenson: *British Journal of Children's Diseases*, April, 1911, p. 145.

In another case a man developed poliomyelitis after a walk of about eight miles. One might ask whether the disease might not have remained in an abortive form if this exertion had been avoided at the onset of the disease. In another case, seen with Dr. W. B. Stewart of Atlantic City, a bite of an insect produced quite a severe sore on the foot, and this was followed in a few days by paralysis. Whether an etiological relation can be attributed to the bite or not is uncertain.

I have been impressed by the large number of adults affected by poliomyelitis in the recent epidemic in this country. During the past winter we had in the nervous dispensary at the University Hospital at the same time three men, aged eighteen, nineteen and twenty-eight years respectively, and one woman in the ward, affected with poliomyelitis; and one woman was in my service at the Philadelphia Hospital at this time with the same disease.

These are the symptoms of an ordinary case of poliomyelitis, but in some instances certain symptoms predominate or develop in a peculiar manner, and thus certain types are established. We may recognize the following types: Abortive; spinal; encephalic, or a combination of this with the spinal type; meningeal; polyneuritic; Landry's paralysis; ataxic; possibly a herpetic; and the late atrophic type.

The abortive type has received recognition only in recent years. It is not likely to be recognized unless an epidemic exists, and at that time probably is very common, but whether cases of this kind may amount to fifty per cent. or more of all the cases, as Wickman[3] believes, seems questionable. The abortive form is considered by some to be more common in adults, and if this be true it may be that adults are more nearly immune. The symptoms do not progress beyond those of the initial stage of the disease, and may easily be overlooked, with the danger of communication of the disease to others. It is not uncommon to find that one member of a family has had a typical case with paralysis, while another has not progressed beyond the initial stage, as in the following cases, in one of which pain was a predominant symptom.

Two children in the same family had severe sore throat the last of August with some eruption over the body. The latter sign is interesting, as eruption has occurred in a certain number of cases of poliomyelitis. The younger child, a boy six years old, had high fever and headache on September 17, 1910. He was not paralyzed and recovered entirely. A week after the symptoms began in the boy, the other child, a girl aged nine years, had severe fever. I saw her on the sixteenth day of her illness. From the onset of the disease she had had severe pain in the lower limbs, which was not checked by morphin. Difficult respiration appeared at the onset of the disease and persisted in increased intensity. The movements of the upper limbs were possible at all parts but were very weak. The lower limbs were almost completely paralyzed and the muscles and nerves of these limbs were tender to slight pressure.

Lumbar puncture was performed and the fluid squirted out, showing considerable pressure. One and a half ounces were removed. The severe pain ceased immediately and breathing became easier. Although entire relief of pain was not obtained by the puncture, the alleviation of this distressing symptom was very marked.

The spinal type is the typical form and is well known. It has been described in the above remarks regarding symptomatology.

The encephalic type is uncommon. The name poliomyelitis is unfortunate in referring to an encephalic type. When symptoms of implication of the brain are present they are usually associated with spinal the limbs and trunk. Fibrillary tremors were present in the left lower limb and both upper limbs.

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[3] Wickman: *Handbuch der Neurologie*, Vol. 2; Specielle Neurologie, 1.

The upper limbs were much atrophied, especially in the forearms and hands, and the grasp of each hand was very feeble. The biceps tendon reflex was exaggerated on each side, but the triceps tendon reflexes were not obtained, and the triceps muscles were much atrophied. The muscles of the shoulder girdle were much wasted.

The tongue was much atrophied. The power of whistling was lost. The condition became progressively worse. Speech became very indistinct, and the man died several years later of an intercurrent disease. A necropsy was not obtained.

DIFFERENTIAL DIAGNOSIS.

In a well-marked case poliomyelitis can hardly escape detection, but in the initial stage an incorrect diagnosis is frequently made. The pain of the limbs may suggest rheumatism, but it is seldom confined to the joints. The onset may be like that of typhoid fever, but the later developments do not substantiate this diagnosis. Influenza on account of the respiratory symptoms, the catarrh and angina, may occur to the diagnostician in the onset of the disease. A beginning palsy in children may readily be overlooked, and failure of patellar reflexes, not always easily determined in children, may be the only objective sign of poliomyelitis.

The resemblance to meningitis may suggest some form of this disorder, as the epidemic or tuberculous. Herpes labialis is common in meningitis, rare in poliomyelitis; psychic symptoms are greater in meningitis, and in poliomyelitis the meningeal symptoms are more spinal than cerebral. The cerebrospinal fluid obtained by lumbar puncture is cloudy and shows leukocytosis in meningitis, and the meningococcus may be found in smear preparations. In tuberculous meningitis the fluid is clear and shows lymphocytosis. Tuberculous meningitis seldom has the acute onset, and the finding of bacilli is of great value. The distinction between the encephalic type of meningitis and encephalitis may be very difficult. Koplik[9] suggests as a point of distinction that in the latter there is a short preliminary period in which the patient, having had a high fever, continues to be about. There is increasing sopor which extends over days, and this is uncommon in meningitis. In poliomyelitis there is usually a more rapid disappearance of severe symptoms of irritation, cranial nerve palsies are more likely to be without fever, and the palsy is almost always flaccid.

In true multiple neuritis the course of the disease is slower, the early febrile stage is absent, the palsy is usually symmetrical and greater in peripheral parts. Poliomyelitis may begin with angina and thus suggest diphtheritic neuritis, but the paralysis follows the angina much later in diphtheritic multiple neuritis. There is not likely to be paralysis of the soft palate and of accommodation, or cardiac symptoms in poliomyelitis, and these are common after diphtheria. Multiple neuritis is believed by some to be more common in children than has been taught.

It is questionable whether myelitis is essentially different in etiology from poliomyelitis. The symptoms differ merely because in the former more of the transverse area of the cord is involved. I have observed myelitis in an adult which developed last year during the epidemic of poliomyelitis, and it has occurred to me that possibly this case was a manifestation of poliomyelitis.

Muscular dystrophy could be considered only in the late stages of poliomyelitis, and at this period confusion is possible. I have seen cases in which the differential diagnosis was difficult.

TREATMENT.

As the possibility of infection through the nasal mucous membrane has been demonstrated experimentally by inhalation of an emulsion of an infected spinal cord, and by rubbing it into the nasal mucous membrane, it is well to disinfect the nasal mucous membrane so far as possible by some antiseptic mouth wash and spray. Hydrogen peroxid or menthol is valuable for nasal disinfection. There is much reason to believe the disease may be transmitted by healthy carriers, and disinfection of the nasopharyngeal passage of persons exposed to the disease is desirable. It is questionable at present whether either food or insects communicate the infection. Breast-fed babies in some epidemics have been chiefly affected.

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[9] Koplik: *American Journal of the Medical Sciences*, June, 1911.

Recovery is not to be despaired of, even when the paralysis has been pronounced. The affected children should be isolated, and other children of the family should be kept from school for at least three weeks. The room, the clothing worn by the patient, and the bedding used should be disinfected. Rest at the onset of the disease is desirable. There is a possibility that some cases may be arrested in the abortive stage by rest. Excessive exertion at this time possibly may bring on a paralysis that might have been avoided. Pain may be treated by antineuralgic remedies, but these may fail to relieve severe pain. I have known lumbar puncture to give great relief under such circumstances. Hot baths for ten or fifteen minutes every four hours, three or four times daily, have been found to lessen pain and produce sleep. Suppositories of opium with extract of belladonna may be useful.

Hexamethylenamin (urotropin) may have some effect in disinfecting the spinal fluid, but if long continued there is a possibility that the formalin set free may have a hardening effect upon the spinal cord. Two grains may be given every two hours during the first two or three days. The drug seems to have an inhibitory action rather than a curative. Calomel is a useful early remedy when constipation is present, and may be combined with soda. It may have some disinfectant action on the bowels, by which the poison is supposed to find one of its means of entrance to the system. Castor oil also is useful.

Pneumonia and bronchitis are to be feared in some cases, and exposure to draught is to be avoided. The danger is increased by paralysis of the respiratory muscles.

Diaphoresis is desirable, and may be obtained by means of hot drinks, packing and hot air. Some simple remedy, as citrate of potassium, is useful for its action on the kidneys.

The tendency to contracture by faulty position and weight of the bed clothing must be avoided. Electricity may be employed after three or four weeks, and where muscles do not respond to the faradic current, the galvanic should be employed. The electricity should only be given by someone familiar with its use. Active movements may be made against resistance to the hand of another person, and attempted movements may be assisted by the electric current.

The serum treatment, according to Flexner, is in the experimental stage, and it can not be predicted how soon or whether at all such a form of specific treatment of the disease will be applicable to the spontaneous epidemic disease in man.

The Histopathology of the Preparalytic Stage of Acute Anterior Poliomyelitis.*

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(Read before the Section on Medicine, Medical Society of the State of Pennsylvania, Harrisburg Session, September 26, 1911.)

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Our knowledge of the histopathological picture of acute anterior poliomyelitis together with its physiological interpretation dates from the work of Prévost and Vulpian who were the first to direct attention to the degeneration and disappearance of the anterior cornual cells in the segments of the spinal cord controlling the paralyzed parts. This work was published in 1866, twenty-six years after that of Heine entitled "Beobachtungen über Lähmungszustände der unteren Extremitäten."

It was Wickman who first suggested the ill-advised name, "Heine-Medinsche Krankheit." It was also Wickman who first advanced the lymphogenic origin of the disease.

Without attempting a résumé of the congested literature on the subject I shall preface my few remarks by a statement of the histopathology of the acute stage of the disease as pictured by Goldscheider and Brasch. This will give us the status of the subject up to and including the year 1900. I shall then speak of the histopathological work of Ivan Wickman, which brings our knowledge up to 1910. After this I shall make a few observations on some experimental work of my own, done at the University of Pennsylvania.

Goldscheider and Brasch say that on gross inspection the spinal cord is somewhat swollen in the region of the affected segments. On cross-section through the affected area the differentiation between gray and white matter is greatly reduced. The gray matter, especially of the anterior horns, appears softened, swollen and of a deep red color. The white substance immediately contiguous to the gray matter presents a reddish tint which nearer the periphery of the cord blends into a grayish red tone.

The microscopical examination of cross sections through the diseased area shows the vessels of the pia mater, especially in the anterior aspect of the cord, greatly distended. The small round-cell infiltration of the pia is inconspicuous. The small vessels of the anterior fissure and the central artery are distended and their walls surrounded and infiltrated with mononuclear round cells. Small hemorrhagic foci, chiefly confined to the gray matter, are of frequent occurrence. The anterior cornual cells at first show a swelling and a rounding of the cell body due to disappearance of the dendritic processes. Disappearance of the nuclei is an early finding. And particularly noteworthy is the fact that the Nissl bodies early undergo a granular disintegration and the cytoplasm stains a homogeneous light blue. Vacuoles are frequently seen.

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These changes are not of necessity limited to the anterior horns but are at times noted in the posterior horns and in the vesicular column of Stilling-Clarke.

The fact that the cells at the periphery of a diseased focus were less involved than those at the center caused Matthes to think that the histogenesis was not parenchymatous in relation to the nerve cell but had its origin rather from the blood vessels which became the centers of the affected areas.

Now this exposition of Goldscheider and Brasch is founded on cases in patients who had died of acute anterior poliomyelitis of two weeks' duration. I wish particularly to emphasize this point as it will have a critical bearing on the histopathological findings of my own work.

The work of Wickman brings the subject down to 1911.

In dealing with the several investigators who have at different times claimed to have found microörganisms supposedly of etiological import, he says that these bacteria have been proved to have nothing to do with the causation of the disease, but either they are due to uncleanly technic or are accidental findings.

Wickman says that in many cases the spleen is enlarged and that inflammatory change is found in the kidneys. He gives other lesions of the abdominal and thoracic viscera which he thinks most likely coincidental.

The cerebrospinal fluid is clear but sometimes increased in amount. The pia at the involved level is hyperemic. In other respects Wickman's macroscopical description corresponds to that of Goldscheider and Brasch.

Microscopically the pia shows a round-cell infiltration mostly of lymphocytic character. There are also polyblasts, plasmazellen, large lymphocytes and fibroblasts, together with typical polymorphonuclear leukocytes. This infiltration is present in the lumbosacral region entirely surrounding the cord, but in the cervical region, chiefly in the anterior aspect. In all cases the infiltration is greater in the anterior than in the posterior aspect. There seems to be no disposition on the part of this infiltration to penetrate the contiguous white matter, it being chiefly in association with the greatly distended blood vessels. No matter how free from infiltration the pial septa at the lateral and posterior aspects of the cord, yet the septum of the anterior fissure is always grossly affected.

The substance of the cord shows great increase in cell content. This is not only the perivascular-cell infiltration such as is seen in the meninges, but also a multiplication of nuclei which on serial section is seen to be independent of a neighboring blood vessel.

The histogenesis of these cells is a most moot question. Whether they are wandering leukocytes, as is held by Schmauss and others, or are proliferated fixed elements, as is held by Goldscheider and Strauss, is yet undetermined.

Wickman calls especial attention to the edema in the cord and suggests that the rapid disappearance of paralytic symptoms may be dependent on a sudden absorption of this fluid.

On May 26 I injected a virus into the brains of seven monkeys. The virus was prepared and furnished to me by my friend, Dr. Paul Lewis, to whom I am grateful for many points in technic. Four cubic centimeters were used in each case as it was thought that there was a probability that the virus was not of great power. The rapid appearance of symptoms is therefore in some measure accounted for.

It was my purpose to kill one monkey every twenty-four hours beginning forty-two hours after the animals were injected. My outline of work comprised the following studies: (1) Posterior ganglia, right brachial plexus; (2) posterior ganglia, left brachial plexus; (3) Bielschowski neurofibril stain of cervical enlargement; (4) hematoxylin-eosin, Nissl and my own azure-eosin methods in the cervical enlargement; (5) a longitudinal study in coronal serial sections through the cervical enlargement stained by the hematoxylin-eosin method. The same scheme of work was to be carried out in the lumbosacral region of the cord.

During my absence in Europe this past summer, through a misunderstanding a large part of the material was not prepared for the microscope. I shall report later a more detailed and complete study.

The monkeys in every case, excepting No. 5, were killed by etherization. Monkey No. 1 was killed May 28, at 10:30 A.M.; No. 2, May 29, at 10:30 A.M.; No. 3, May 30, at 10:30 A.M.; No. 4, May 30, at 1 P.M.; No. 5 died during the night of May 30-31, and was placed in the refrigerating apparatus by my *Diener*. Monkey No. 6 was killed May 31, at 10:30 A.M. Monkey No. 7 was etherized May 31, at 4 P.M. and his heart activity injected with an aqueous solution of azure II. He became intensely blue within ten seconds and, after a general clonic convulsion, of about half a minute in duration, died.

The first symptom of paralysis was noted on the morning of May 30 in one monkey which was killed at 10:30 that morning and labeled No. 3. There was some voluntary motion remaining in his right lower limb, but the grasp of his right foot was decidedly weak. Each of the other limbs was the seat of a complete flaccid paralysis. The breathing mechanism and the muscles of the neck and back appeared normal.

At the time monkey No. 3 was killed, monkey No. 4 was running all over the cage using all the extremities but the right upper limb, which hung limp at his side. At 1 P.M. of the same day both upper limbs hung limp, but there was still power in the left upper limb which could be elevated above his head if it was stuck with a pin. The legs were uninvolved and strong at the time of death.

Monkey No. 5 began to show weakness in the upper limbs the morning of May 30, and a note made at 9 A.M. of this date states that although the animal could get around the cage with some agility yet the movements were distinctly awkward. By 10 P.M., May 30, there was almost complete flaccid paralysis of all four extremities and the muscles of the neck and the erector spinæ. The animal died during the night.

At 9 P.M., May 30, the note was made that monkey No. 6 showed total paralysis of right upper extremity and an awkwardness in the lower extremities in moving about. At 9 A.M., May 31, there was noted general weakness of both lower extremities and left upper extremity. The muscles of the neck and the erector spinæ were well preserved.

The VonLenhossék-Nissl preparations from the cervical and lumbar enlargements of monkey No. 6 show all stages of motor nerve-cell disintegration. There are very few cells which can be said to approach the normal. The same remark holds good for the lumbosacral region.

From my incomplete study I should say that one of the very early pictures in this nerve-cell degeneration is a tendency for the cytoplasm to stain homogeneously. The tigroid substance becomes finely granular and evenly dispersed and later disappears altogether. The eccentric placing of the nucleus was not a frequent finding in my sections, but the flattening and deformity of contour was of common occurrence. A few groups of cells were found in which there was homogeneous staining of cytoplasm, enlargement of nucleus with disappearance of nucleolus and a very marked, highly refractive nuclear periphery.

Israel Strauss, in his scholarly study of the histopathology, remarks the disappearance of the neurofibrils as one of the earliest pictures. My Bielschowski preparations confirmed this but also showed another interesting point, to wit: In a number of instances I have found a group of anterior cornual nerve cells showing normal neurofibrillar structure when all the other groups in the anterior horn had lost their neurofibrils. It is a finding of this kind which accentuates the blood-vessel genesis of this disease.

I shall not detail the pictures of neuronophagia which I have found correspond to the descriptions of Strauss, Cadwalader, Wickman, Goldscheider and many others.

In careful search through serial sections it was possible in almost every case to trace a blood vessel to each focus of disease. And where this could not be done, the sections at either the upper or lower level of the focus were missing.

In only a few instances did I find Fettkörnchenzellen. Probably had the process been allowed to run on a few days longer these fatty cells would have appeared.

I was not able to mark any distinction between the perivascular infiltration of veins and arteries, such as has been described by some and denied by others.

I was much interested in the small round-cell infiltration of the pia. Some observers, Wickman, for example, mention the fact that this infiltration completely surrounds the cord in many cases in the lumbosacral region but is inconspicuous in the cervical enlargement, and when present confined chiefly to the anterior aspect. This was precisely what I found in the monkeys examined.

Now when you realize that the paralysis in my cases was much more pronounced and early in the upper limbs than in the lower limbs, it would argue that the meningitis is not an active factor in the development of the symptom picture, with the single possible and questionable exception of the presence of pain.

I think that the possibility of this difference in degree of small round-cell infiltration in cervical and lumbosacral regions being due to a gravity condition should claim critical thought.

In no section which I examined did I find any free hemorrhagic foci, minute or otherwise. The gross distention of blood vessels and the areas of marked edema were of course of frequent occurrence.

The posterior ganglia and roots showed no marked peculiarity. In one posterior root I detected a varicose and swollen condition of the medullary sheaths of a few nerve fibers, but this has been remarked before by Wickman and others.

In closing I would call attention to the two theories of the development of this disease which have direct bearing on histogenesis; that of Charcot, who stated that the primary toxic injury was located in the ganglion cell and that the replacement with glia elements was secondary, and that of Erb, who held that the disease was an inflammatory, exudative process with secondary involvement of the nerve cells. Erb's theory certainly claims the majority of support to-day, and Wickman's theory, stated in the early part of this paper, seems to me at the best an amendment to Erb.

I shall not go into the subject of the cytology of anterior poliomyelitis. Marschalko or Aschoff may best be consulted by any one desiring the theories of histogenesis of the large number of cell forms described.

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SOME RECENT CLINICAL INVESTIGATIONS OF POLIOMYELITIS *

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Since June of the present year (1910) I have had the opportunity of studying about forty-five cases of poliomyelitis, and of course have had the usual experience of the neurologist with the disease for many years, this including the observation of cases extending into the hundreds. What I have to say in this paper about some special features in symptomatology, diagnosis, and treatment, is in the main based upon my recent experience.

A discussion of the subject naturally divides itself into a consideration of the acute and residual manifestations.

ABORTIVE CASES

A considerable number of facts came to my knowledge while studying the disease at Bethlehem and elsewhere in the Lehigh Valley, and also in Philadelphia in the ordinary course of my work during the last few months, which seem to indicate that instances of the disease of a mild or abortive type occurred and were frequently overlooked. Children in the same neighborhood and sometimes in the same family as those suffering from well-marked acute and residual phenomena of the disease were reported to others and myself as suffering from slight fever, malaise with digestive or other symptoms and sometimes with a passing weakness of one or more limbs. Such cases have importance in a general

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* A paper read at a special meeting of the College of Physicians of Philadelphia, held October 14, 1910, for the discussion of poliomyelitis, especially with reference to its study during the recent epidemic in the Lehigh Valley, Pennsylvania, and elsewhere.

study of an endemic or epidemic, as showing that the infection of the disease may be more widespread in its influence than appears from a knowledge of the disease as illustrated by well-defined examples. Some of those who have discussed the epidemics occurring in different parts of this country, as well as in Norway and elsewhere abroad, have directed attention to this matter of abortive types of poliomyelitis.

FULMINANT OR RAPIDLY FATAL CASES

In Bethlehem I received information regarding three cases of fever which were rapidly fatal and which probably were examples of poliomyelitis. The diagnosis of these cases, owing to their speedy termination, could not be fully made. The children were under three years of age and died within forty-eight hours of the onset of high fever, with delirium and convulsions, with in two instances retraction of the head and digestive disturbances. Death apparently resulted from the overwhelming influence of some toxæmia.

In Philadelphia a healthy, strong young adult, who had been quite well, was taken with feelings of weakness and general distress for which he could not account, and in less than two days, although he still remained on his feet, he was found to have some fever and then went to bed. Some loss of power was evident in his upper extremities, although the gravity of his condition was not fully recognized. His mind remained clear. He rapidly sank and died within four days after the recognition of the first symptoms of his disease.

In another case, that of a man between thirty and forty years of age, with high fever, the patient's condition pointed to poliomyelitis and bulbar encephalitis. He died within a week of the onset, paralysis having appeared before death in the limbs of both sides and in the domain of the facial, glossopharyngeal and other cranial nerves. Paralysis in this case was chiefly of the flaccid type.

Through the courtesy of Dr. M. Howard Fussel I obtained a necropsy on one fulminant case, that of a child about seven years of age, who died four to five days after the onset of fever. The paralysis was of both the bulbar and spinal type, this being more marked in the upper extremities and cranial nerves than in the lower extremities.

A miscroscopic examination was made in this case by Dr. E. M. Williams, of the clinical and pathological staff of the neurological department of the University of Pennsylvania, who has furnished me with the following preliminary report upon his findings: Macroscopically no change was visible other than a congestion in the meninges, with some irregularity in the shape of the cord in the cervical region. Sections were made from the rolandic area in the cortex, the cerebellum, the oblongata, and the lower cervical, mid-dorsal, and lumbar regions of the cord. Section of the cortex showed a general weakness in the staining qualities of the chromophile bodies in the cells, there being a diffuse pale staining of the entire cell. The nuclei were indistinct but no evidence of neuronophagocytosis was present. There were a few very small areas of beginning softening.

The meninges were very tensely congested and somewhat infiltrated, being of a lymphocytic character. Few leucocytes were present. Many cells resembling in size and shape the so-called plasma cells were found throughout the pia. Extensions of the pia into the substance of the brain were also greatly infiltrated and congested. There was no distinct infiltration of the substance itself.

The cellular changes above noted might have been either toxæmic or *post mortem,* as there was no sign of degeneration other than the chromolysis above mentioned, the nuclei being centrally placed with very few exceptions.

The cerebellar cortex as well as sections of the dentate nucleus showed no pathological changes.

In sections of the oblongata intense infiltration, both around and in the vessel walls, was present, and the vessels were very greatly congested. In some areas were clumps of round cells resembling in appearance abscess formation, in these cases round cells taking the place of polymorphonuclear leucocytes. Fairly numerous areas of softening occurred throughout the section, in many cases with hemorrhagic extravasation. The cellular degeneration was very marked, particularly in the posterior or dorsal part of the oblongata. The cells forming the ninth, tenth, and eleventh nuclei were greatly affected. The nuclei of these cells were often somewhat blurred. The cells also of the inferior olive of the lateral nuclei were somewhat degenerated. In contrast to these the nucleus of the twelfth stood out distinctly and clearly with well-stained tigroid bodies and distinct nuclei. In only a few cells was there any sign of disintegration seen, this being shown by slight lessening in the number and clear staining qualities of the tigroid bodies.

The sections of the cervical segment showed the meninges decidedly congested and infiltrated, the infiltration being of a round cell character. The ventral part of the cord was especially involved, the vessels here being swollen and congested, and the membranes apparently thickened. The anterior roots showed a swelling of the medullary sheaths. Throughout the entire section was complete destruction of the cellular element of the gray matter, the only remnant of the cells being an occasional, irregularly shaped nucleus. The lymphocytic infiltration was intense, particularly throughout the gray matter. It was most marked in the coats of the vessels and also extended throughout the surrounding tissue. Many areas of softening were present in the white matter. This was marked in the anterior and anterolateral parts of the cord near the periphery. In some of these areas was total destruction of the fibres. In others, where the softening was apparently just beginning, the myelin sheaths were swollen and œdematous and the axis cylinders were irregular in shape, laterally displaced, sometimes swollen and even broken up.

Sections in the mid-dorsal region also showed intense infiltrative changes with thickening of the pia, but the infiltration was not so great as in the cervical region, while the cells in the anterior horns in this section were all destroyed. A great many cells of the posterior horns were also slightly degenerated, some of these in Clarke's column and posterior and lateral to Clarke's column, still showed well staining chromophile bodies and distinctly outlined nuclei. Others in these groups had peripherally arranged tigroid bodies. Even these cells had not the clear cut, regular chromophile bodies such as those found in normal cells, and were probably somewhat affected by the toxæmic condition.

The lumbar region showed thickening of the pia with marked round cell infiltration, this being particularly noticeable in the anterior part, where the vessels were especially congested and infiltrated. These cellular substances showed the same great degree of infiltration with an absence of all posterior horn cells. The anterior cells, however, were only in a few instances totally destroyed. Most of them had normally placed nuclei, although the chromophile bodies stained very poorly and there was probably a certain degree of toxæmic change present.

VARIETIES IN ONSET AND COURSE

In several of the cases of poliomyelitis observed by me during the recent epidemic the symptoms usually present at the onset of the disease were absent, or what is more probable, were overlooked. The parents and family were unaware of any febrile, gastro-intestinal, or nervous symptoms, their attention having been first directed to the disease by the awkwardness or absence of movements in the affected parts.

The onset and acute stage of the disease were usually after the fashion described in the text-books, namely the sudden beginning with fever, the paralysis being observed in from two to four days after the first febrile manifestations, this increasing steadily to a maximum and then regressing.

In two instances the preparalytic stage was long, the paralysis not appearing until the second week.

In three instances the disease assumed a relapsing or recrudescing type. In all of these the musculature of one part of the body was attacked. The fever disappeared, and with its reappearance another part became the seat of the paralysis. In these cases the lower extremities were commonly first affected, the upper later. In one, bulbar and cerebral symptoms appeared and death resulted.

In this case the patient was a boy thirteen years of age, who after a high fever was paralyzed in both upper and lower extremities, the abdominal and thoracic muscles, the diaphragm escaping. Some paralysis of the muscles of articulation was present, and the left abducens was paretic, the patient having diplopia. On the tenth and eleventh day after the onset, the temperature had returned to normal and the stage of regression seemed to have begun, but two days later the temperature again began to rise and after one or two oscillations remained high, paralysis of respiration set in, and death ensued.

The second recrudescing case was that of a boy about one year old. Early in July he had pain in his lower extremities, and in a day or two was paralyzed in them. When first seen by me on September 12th he was partially paralyzed in both lower extremities, more on the right than on the left, and most markedly in the peroneal muscles, although there was some impairment of power in flexing and extending the thighs. Ten days after this examination the child had some fever and general distress, and was found to be paralyzed in both upper extremities, most marked proximally. He also had difficulty in holding his head erect, apparently from paralysis of the muscles of the neck.

PERIOD OF INCUBATION

The period of incubation of poliomyelitis is a matter of some interest and one which seems to have received but little attention from writers on the subject. It is difficult to get facts of value bearing upon this question, but a few observations indicate that this period ranged between a week and three weeks. In the majority of cases it is probably from one to two weeks.

Two boys aged respectively nine and thirteen years, were seized with the disease while on an island on the Virginia coast, they having come from Bethlehem, in one instance one week and in the other nine days before the first symptoms of the acute stage of the disease appeared. As no other case appeared among the children, about thirty in number, who were present in the same camp, this observation seemed to indicate that the period of incubation was at least one to two weeks in length. In another case seen by me at Stroudsburg, the only instance of the disease in this city up to the time of my visit, the patient, a boy of about ten years of age, had been with other members of his family in the Connecticut Valley at a point about ten miles from Springfield, where the disease as is well known exhibited great virulence, the endemic there having had many serious and even fatal cases. In one case seen at Bethlehem the child had gone from one portion of the borough where the disease had appeared in others, to another which was relatively free, the symptoms of poliomyelitis appearing about a week after the change of location. In two other cases the evidence seemed clear that infection had taken place a little less than two weeks before the change of location.

CLINICAL TYPES ACCORDING TO THE LOCATION OF THE LESION AND RESIDUAL PARALYSIS

It is not my intention, either from recent personal experience or from a study of literature, to present statistics regarding poliomyelitis, but rather to concentrate the attention of others, as my own attention has been concentrated, on a few salient features of the disease as shown in the cases recently observed. The statistics of value regarding the occurrence and special features of poliomyelitis, are those which are now being presented in current journals, in reports of medical associations and of State, county and municipal boards of health. In the main these agree as to the age most frequently attacked being below three or four years, as to the most virulent months of prevalence of the disease being July, August and September, as to the disease being most commonly rural, as to the residual paralysis being usually in one or both lower extremities, as to the existence of some fulminant and many abortive cases, as to the usually serious significance of symptoms showing implication of bulbar centres, and as to the probable infective nature of the disease. My own recent experience is in accordance with these well known determinations.

All but five of the forty-five cases were instances of the disease which have occurred since May of the present year. The others, although studied during recent months, were left paralyzed by previous attacks, these however, having been within two or three years.

A classification of these cases, chiefly according to the location of the lesion and the residual paralysis cannot be thoroughly made, but an abstract statement of them may be given.

Five cases belong to the fulminant type to which reference has been made; five were abortive cases; eight were cases of paralysis in one lower extremity; fifteen of paralysis in both lower extremities; twelve in four or three extremities, usually four; in one case the paralysis was confined to the upper extremities; one was a case of uncomplicated bulbar paralysis (facial); in five cases paralysis of the extremities was associated with bulbar paralysis.

In the majority of cases of paralysis involving both upper and lower extremities the abdominal and other truncal muscles were implicated early. The abdominal paralysis persisted in several instances. The bladder escaped except in two or three cases. Constipation, which may have been due in some of the instances at least to a paralyzed condition of the bowels, was the rule, diarrhœa being present only in a few cases, although my data on this matter are not full. In the fulminant cases both bulbar and cerebral centres (cortex and basal ganglia) were, from the symptomatology, probably involved. In the one case with necropsy this wide-spread implication was determined.

While the assault of bulbar centres is always of serious significance it was nevertheless shown by a study of these cases that cranial nerves like the facial, might be the seat of residual paralysis in patients who make good or comparatively good general recoveries.

In five cases the disease occurred in adults, three of these being between the ages of twenty and thirty years, one between thirty and forty, and one between forty and fifty years.

In the cases recently seen I have not observed any well-defined instance of paralysis seemingly due to Strumpel's circumscribed cortical encephalitis. I can see no reason, however, why such cases may not occur and a few have been recorded. In the fulminant cases to which attention has been directed, in fact all parts of the brain as well as the bulbar and spinal portions of the nervous system, are doubtless often the seat of inflammation. In cases with necropsy and microscopic examination, studied in connection with epidemics of poliomyelitis which have occurred especially in Norway and Sweden and in this country, lesions, practically identical with those in the cord have been found not only in the cortex but also in the basal ganglia. In most of these cases the effects of the inflammation upon the encephalitic tissues have not been sufficient to bring about permanent residual palsies like those left by the cornual inflammation. The toxæmia, in other words, has been more destructive to cord centres than to those of the higher regions of the nervous system. Cases have been observed, however, in which a combination of cortical paralysis has been associated with evidences of spinal atrophic paralysis or of bulbar atrophic paralysis.

Cases with symptoms of onset like poliomyelitis and with the so-called cerebello-rubro-spinal symptomatology—coarse tremor, with some paresis and rigidity, the whole somewhat like paralysis agitans or a mixture of this disease and multiple sclerosis—have also not been observed, although in one child some ataxia was present.

The comparative frequency with which the upper as well as the lower extremities were left paralyzed is of much interest and shows the seriousness of the infection. The residual paralysis in the upper extremities was usually much more decidedly proximal than distal, the movements of the shoulder and upper arm suffering most. These observations as to the frequency and severity in the epidemic, of cervical lesions are in accordance with those made by some other recent observers.

DIAGNOSIS

While the diagnosis of poliomyelitis is usually not difficult, mistakes are not infrequently made in the early days of the disease. In the period of residual paralysis, the differential features of the disease are now so well known as scarcely to need discussion. They are chiefly the presence of flaccid paralysis, often localized to parts of one or more limbs, with decreased reflexes, atrophy, vasomotor changes, lost faradic irritability and changes in galvanic reactions, secondary contractures and deformities, the absence of sensory phenomena, the rarity of paralytic implication of the bladder, and the sparing of the cerebral functions and faculties. The diagnosis occasionally needs some consideration when the paralysis chances to attack an arm and leg on one side, as is the case commonly in the cerebral palsies of children, but the residual phenomena are here so radically different as not to cause much doubt or confusion. In the cerebral cases spasticity instead of flaccidity, increased instead of decreased reflexes, retained response to the electrical currents, and the presence of other evidences of cerebral involvement, including even the history of an apoplectiform and convulsive onset, will assist in the easy differentiation.

A knowledge of the existence of an endemic or epidemic of poliomyelitis is of course an aid to diagnosis, but may occasionally be misleading, most probably in the direction of an erroneous diagnosis of the disease when a child is attacked with fever due to digestive disorders or with an exanthematous affection.

At the onset there is very little to distinguish the fever of poliomyelitis from that of any other infectious or contagious disease of childhood. Of course in the case of exanthemata the appearance of the distinctive eruption identifies the disease, but this help is not available in the case of diseases like typhoid and malarial fevers. Gastro-intestinal irritation,—vomiting and diarrhœa,—is frequent in the acute stage of poliomyelitis, and yet constipation was the rule in the cases observed by me in the present epidemic. The occurrence of cases similarly affected in the same locality and especially in the same family, always leads to the suspicion of the existence of the disease. The fever of poliomyelitis presents so many variations in its intensity as to be misleading. The severity of the phenomena in the febrile stage appears to be to a considerable extent dependent upon the degree to which the cord is invaded. A case in which the entire spinal cord and even higher levels are toxæmic as a result of the peculiar infection of poliomyelitis, gives fever of high grade. In a severe case of poliomyelitis a study of the musculature of the thorax and abdomen, even during the first days of the disease may be of some advantage. The abdominal and thoracic walls become, if not paralyzed, at least partially so in a considerable number of cases, facts which are evidenced by peculiar breathing, and by relaxation of the abdominal walls.

The deep reflexes may be affected very early before paralysis is suspected. It follows especially that in every case of acute fever, obscure in its nature, occurring in children or youths, the reflexes should be thoroughly investigated.

At the very beginning of the disease, as is also true with multiple neuritis in children or adults, the knee-jerk and perhaps other deep reflexes may be increased, but this soon gives way to decrease or loss.

The point of importance is, that while it is impossible to give any rule as to the time of the coming on of the paralysis and diminished reflexes, these usually occur before, sometimes considerably before, the physician recognizes their presence. In no small number of cases recently studied, the paralysis and impaired reflexes were overlooked for some days.

Dr. Herbert Fox of the Pennsylvania State Department of Health suggests the value of a blood examination in abortive or other doubtful cases, and especially the diagnostic importance of an excess of lymphocytes, and of the eosinophile percentage.

The erroneous diagnosis most frequently made during the acute stage of poliomyelitis at a time when the disease is prevalent is undoubtedly meningitis. This may be believed by the physician to be the epidemic form of cerebrospinal meningitis, or the diagnosis of meningitis is made without any definite idea as to its character or infectious nature. This mistake is due in some instances to the fact that the patient presents for a short time clinical phenomena such as convulsion, retraction of the head, delirium, and pain or hyperæsthesia in the back or limbs, which are most readily explained on the supposition of a meningitis. Indeed, as necropsies have shown, some involvement of the meninges not infrequently occurs and is the best explanation of such symptoms. In the case studied for me by Dr. Williams, the pia both of the cord and of the brain was congested and infiltrated. The inflammation of the membranes, however, is different in type and intensity from that which is present in true epidemic or sporadic meningitis. The severe, persistent, and destructive effects of the toxin of poliomyelitis fall upon the substance of the cord and brain, when the latter is attacked, and especially upon its gray matter. The meningeal symptoms therefore, while they may in some cases be present are usually transient. A little time is all that is necessary to enable the physician to determine the preponderance of a non-meningeal symptomatology. In the majority of cases no true meningeal symptoms are present.

Another disease of the nervous system from which poliomyelitis must occasionally be separated even in the acute or subacute stage is neuritis. Some writers go so far as to speak of a polyneuritic form of poliomyelitis, making such a subdivision because of their belief that the disease is frequently complicated with a true multiple neuritis. While pain on pressure or manipulation over muscle masses and nerves in certain regions is sometimes present during the first week or two of acute poliomyelitis, this is comparatively rare, at least in my experience. Even when pain in the back or limbs is complained of, this is by no means always due either to meningitis or neuritis, although it may have either of these origins in a limited number of cases. I paid particular attention to the presence and character of the pain in some of the cases recently investigated.

In some of the articles on poliomyelitis in text-books it is set down that pain is to be looked for almost as a matter of course in the head, back, limbs, or trunk. In a considerable majority of cases which I have studied, however, pain was absent, if the reports of the physicians, the parents, and members of the family who had the patient under observation could be depended upon. In others, however, pain was a marked feature. Usually it disappeared after two, three, or four days. The pain was variously located.

One patient, a boy of nineteen years, who gave a very clear account of his early symptoms, said that pain was present in the head, neck, and shoulders, also for a short time in both lower extremities, and in this case the paralysis in the arms and neck and some weakness in the lower extremities came on while the patient was on his feet, and the case was diagnosticated by a physician who saw him as walking typhoid fever. He described the pain as having been of a very severe and worrisome character, especially that which he suffered in his shoulders and upper arms, these being the regions in which the residual paralysis was most marked. Paralysis in the legs was in fact never positively present, although both knee-jerks were absent. In another case in which some of the muscles of the neck were temporarily paralyzed, the patient complained of pain in this region.

I am satisfied that in many instances in which handling the little patients seemed to cause pain, the manifestations were simply evidences of general distress, restlessness, and apprehension, were in other words psychic.

Tenderness in long nerve trunks and in muscle masses, such as is so frequently present in multiple neuritis, was usually absent. In two cases, however, it was present for seven or eight days from the time of onset.

It must not be overlooked that now and then a case of multiple neuritis is observed during the presence of epidemic poliomyelitis. I have seen one remarkable instance of this sort in an adult who had suffered in infancy with poliomyelitis possibly pointing to the fact that although this patient, because of his attack of many years previous, was immune from poliomyelitis, the infection may have taken the form of a multiple neuritis in his case.

Some of the pain which is complained of by patients during the early period after the disappearance of acute symptoms, say during the second, third, and fourth weeks of the disease, is apparently due to the unusual position and dragging of the limbs, these causing great distress which the patient commonly refers to the joints. This was very marked in three cases in and around the knee-joint on one side, although in two of the three the paralysis was wide-spread in both extremities. Two of these patients complained bitterly when the limb was handled and required it to be placed in a position of flexion of the leg almost at right angles to the thigh in order to get relief. Tenderness due to pressure on muscle or nerve trunks was not present in these cases. The handling seemed to cause distress through the production of certain malpositions of the limb,—malpositions at least as regards the patient's consciousness. In these cases, after the lapse of two or three weeks, the pain disappeared, and the limb could be handled with impunity.

TREATMENT

In concluding this paper I shall say a few words about treatment, although unfortunately I have nothing much that is new to present. Generally I have used or have suggested the therapeutic measures indicated in what follows.

As the nature of the micro-organism or toxin producing poliomyelitis is still unknown, no treatment directly applicable to the pathologic cause of the disease can be used. Treatment with our present lights should be directed in the first place to the febrile phenomena and to possible elimination through the skin and gastro-intestinal tract. Fever mixtures containing diaphoretics, and sometimes combined with moderate amounts of bromide to relieve cortical irritability should be used. Sponge bathing with cool or tepid water is of service and the patient should be made, if possible, to drink freely of pure water. The hot pack, which has been recommended by various writers on the subject, can be used with much benefit and is especially valuable in serious cases with high fever.

The bowels should be opened, preferably with small doses of calomel, followed by a moderate saline or castor oil. Especially when constipation is present, and this has been the rule in the cases recently coming under personal observation, the laxatives should be used daily, although not in large doses. An enema of oil, or of this with soap and water, can be resorted to when difficulty is experienced in opening the bowels.

Ergot has had some advocates and may be tried, most efficiently either by the administration of a good fluid extract or by the hypodermic injection of some preparation like ergot antiseptic. The dose used when given by the mouth varies with the age of the patient, ranging usually between ten and thirty minims. On the whole I have not seen much benefit from administering ergot, although it has some considerations in its favor.

Counter irritation, either in the form of extreme heat or cold or wet cups, has not proved of much value. The so-called Bier's method of using large cups retained for a long time in position along the spine, is theoretically a mode of treatment to be approved. The cups should be kept on for long periods with shorter periods off, say for instance two hours on, and one off, this treatment being kept up for several days. It is probably effective, if at all, not in producing anæmia of the cord but in stimulating the activity of absorption. Some difficulty is experienced in using these large cups on children. They were used under my direction in two cases, and in one of these cases, a boy of about nine years of age, with apparent benefit, though I could not be certain of the amount of good that was accomplished through the method, as other measures were used.

The patient should be kept as quiet as possible, and not in one position all the time. It is especially desirable not to allow him to be continuously in the supine position, although probably not as much is accomplished by the prone position or position on the side as is sometimes believed.

Urotropine, because of its known action in cases of meningitis, has something in its favor, and might be used in small doses for a time. One or two grains can be given every two or three hours to small children and as much as five or more to other patients.

The use of mercury in small doses as the acute stage is subsiding may be of some service. Salicylates have been advocated, especially when pain is present, but are of doubtful value. They are probably of advantage when real neuritis is present. When pain and hyperæsthesia are present in the limbs application of hot water for a few minutes to the painful regions can be used and the parts between these applications can be wrapped in rubber dam or in light warm flannels. The painful parts can also be suspended in warm water as has been recommended.

The diet should be light and unstimulating during the febrile stage. The bladder should be catheterized if the urine cannot be passed, although fortunately this procedure is not often necessary.

My experience with lumbar puncture has been very limited, but it is desirable to resort to it for therapeutic reasons in the more severe cases. Some benefit from its use has been observed by others, especially in cases in which the symptoms of the acute stage are serious or threatening.

When paralysis of respiration is threatened through involvement of bulbar centres, oxygen inhalations may be tried and also the hypodermic use of remedies such as atropine, strychnine and nitroglycerin. In one of my cases the most strenuous efforts were made to maintain respiration and cardiac action by such measures but without avail.

Efforts to treat the paralyzed limbs electrically should not be begun too early, although for the purpose of assisting the prognosis a few tests may be carefully made at the end of the first or second week. After all the acute symptoms have subsided, and the residual paralysis begins to be evident, treatment with massage, systematic movements, and electricity can be begun. It is best to begin first with gentle massage and passive movements, reserving the electrical treatment for a week or two later. The faradaic current may be used for those muscles which respond, although imperfectly, but sooner or later the galvanic current slowly interrupted will be found to be the most desirable. Treatment with massage, movements, and electricity should not be continued too long. A good plan is to use it for a period, then stopping for another period to return to its use. Two or three weeks of use and a one or two weeks' interval without treatment is a good rule to pursue for the first six months or a year. Tenotomies to give better position to the limbs, and the use of light apparatus to assist the muscles which retain their power, can be used to advantage, but should always be resorted to with great thoughtfulness and after very careful investigation of all the facts in the case.

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AN ANATOMIC STUDY OF THE FASCICULUS OCCIPITO-FRONTALIS AND THE TAPETUM[1]

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The exact anatomic relations of the fasciculus occipito-frontalis has been the subject of much controversy since Onufrowicz (1) first described in 1887 in the brain of a microcephalic idiot in which the corpus callosum was lacking, the presence of a large fronto-occipital association bundle. A similar finding was afterwards described by Hochhaus (2), Kaufmann (3) and Forel (4).

Dejerine (5) identified this bundle with one which he described in the normal brain as extending along the external angle of the lateral ventricle, within the corona radiata, above the caudate nucleus and beneath and external to the corpus callosum, and between the cingulum and the superior longitudinal bundle of Burdach. It is separated from the ventricular cavity by the subependymal gray matter.

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[1] Read by title at the thirty-sixth annual meeting of the American Neurological Association held in Washington, May 2, 3 and 4, 1910.

It measures one half centimeter in thickness in verticotransverse sections and is pyriform in shape. Clearly defined at the level of the head and trunk of the caudate nucleus it becomes less so at the tail of the latter. Posteriorly these fibers spread out upon the inferior and external wall of the sphenoidal horn forming the tapetum.

This bundle originates in the cortex of the frontal lobe, receiving fibers from the superior and external portion of the hemisphere and enters into the external capsule, crossing probably the fibers of the forceps major.

The fasciculus occipito-frontalis degenerates after lesions of the occipital lobe, according to Dejerine, and he believed that it corresponds to the "couronne rayonnante du noyau caudé" of Meynert and to the "faisceau du corps calleux se rendant à la capsule interne" of Wernicke.

These fibers, he states, are not identical with the "faisceau longitudinal supérieur" (arqué de Burdach) as maintained by Forel and Onufrowicz as well as Kaufmann and Hochhaus, for Dejerine claims that, while the faisceau longitudinalis superioris is situated external to the corona radiata, the "faisceau occipito-frontal" is situated on the contrary internal to the corona radiata and forms a sort of a vault to the lateral ventricle.

According to Meynert (in Dejerine) the "couronne rayonnante du noyau caudé" is composed of fibers which, originating in the caudate nucleus, proceed then to the convolutions of the superior part of the hemisphere. But Dejerine, with Wernicke, did not believe that any of these fibers terminate in the caudate nucleus. Wernicke (in Dejerine) believed however, that these fibers constitute the "faisceau du corps calleux se rendant à la capsule interne" (Balkenbündel zur inneren Kapsel) arising from the knee of the corpus callosum and the white matter of the frontal lobe. These fibers form a compact bundle, measuring one and one half centimeters in thickness, and extend along the superior external border of the caudate nucleus, the fibers penetrating into the internal capsule between the caudate nucleus and the superior border of the putamen at the level of the middle part of the optic thalamus. But Dejerine did not accept this as proved and was of the opinion that this bundle can be identified with the fasciculus occipito-frontalis described by Forel and Onufrowicz in cases of agenesis of the corpus callosum.

Sachs (6) who examined the specimens of Kaufmann was convinced that the latter's case was not one in which the corpus

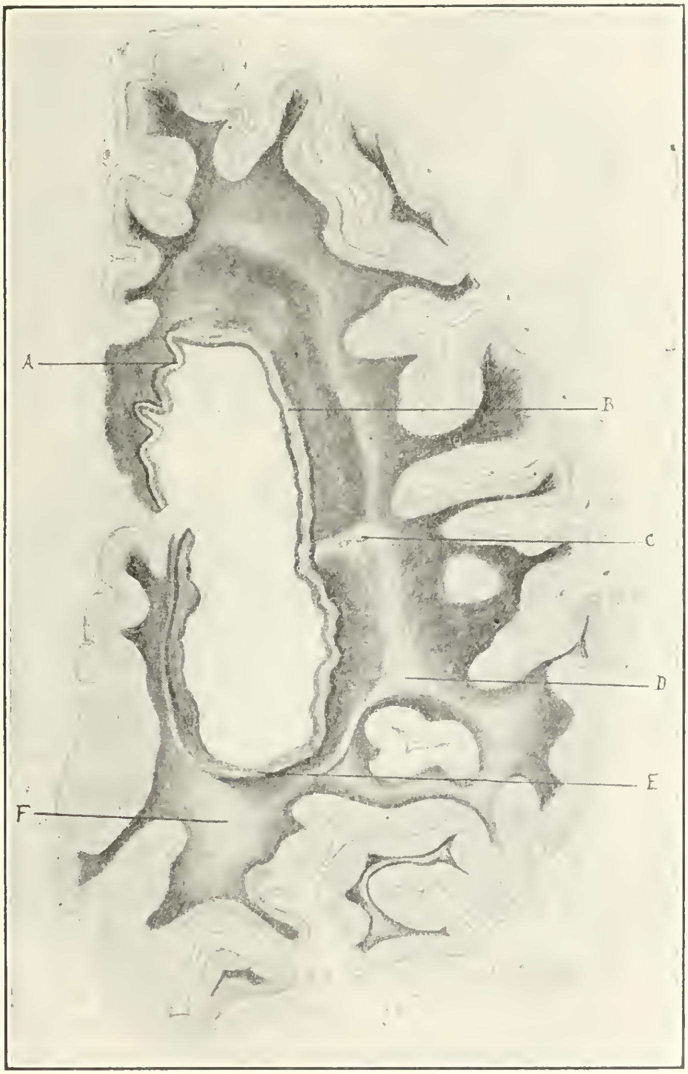

Fig. 1. (Right side.) *A*, area of degeneration which appears to be continuous with the degenerated fasciculus occipito-frontalis at *B* which can be traced around the ventricle; *C*, area of softening; *D*, degenerated white matter at the base of the second parietal convolution; *E*, area of degeneration continuous with *A* and *B*; *F*, degeneration of white matter.

callosum was lacking. He believed that the fibers were all present but did not extend from one side of the brain to the other. It was in the nature of heterotopia and had therefore no important bearing on the subject.

Marchand (7) too believed that the fasciculus occipito-frontalis in agenesis of the corpus callosum did not correspond to a normal bundle in that situation but rather to the fibers which existed normally in the corpus callosum.

Obersteiner and Redlich (8), Schröder (9), Bischoff (10), Römer (11) and Probst (12) also looked upon this bundle as being abnormally situated fibers belonging to the corpus callosum.

While Römer described a bundle of fibers corresponding to the fasciculus occipito-frontalis in horizontal section he was unable to trace a long association bundle in sagittal sections similar to this bundle of fibers and concluded that the findings of Onufrowicz, Kaufmann and others forming the basis for Dejerine's claim for the "faisceau occipito-frontal" were insufficient.

Von Monakow (13) described a bundle similar to the "faisceau occipito-frontal" of Dejerine, and the "reticulo-cortico-Bundle" of Obersteiner and Redlich. The latter authorities proposed the name of "reticulirtes cortico-caudales Bündel" which collects projection fibers from the cortex to the caudate nucleus and which continues as a small bundle into the inferior horn of the lateral ventricle. This bundle consists of large fibers situated somewhat in front of the caudate nucleus and extends backwards lateral to the caudate nucleus.

Zingerle (14) claimed to have demonstrated in his case in which the corpus callosum was only present in its anterior portion corresponding to the knee of the corpus callosum, the presence of a long association system between the frontal, parietal and occipital regions, analogous to the fronto-occipital bundle of Onufrowicz and the fasciculus subcallosus of Muratow. Schröder did not believe that this case was analogous to those of Kaufmann, Onufrowicz and Hochhaus, which were cases of defective development while that of Zingerle was one of softening and atrophy.

As the result of experiments upon animals, Muratow (15) concluded that the fasciculus occipito-frontalis of Onufrowicz and Kaufmann, called by Muratow the fasciculus sub-callosus, is a long association bundle which degenerates in the same hemisphere in which the frontal or occipital lobe has been destroyed. After the corpus callosum was cut in his animals, there was no degeneration found in the tapetum nor the fasciculus subcallosus. He did not believe that in the fasciculus subcallosus, the fibers extended throughout the entire system.

Obersteiner and Redlich, as the result of experiments, concluded that the fasciculus subcallosus, which, in their opinion was identical with the " faisceau nuclei caudati " of Sachs, had nothing whatever to do with the fasciculus occipito-frontalis of Onufrowicz, but that this bundle consisted of fibers of the corpus callosum in all probability.

The fasciculus occipito-frontalis, as described by Dejerine, Obersteiner and Redlich, is recognized as a narrow bundle that can be differentiated in man but not in animals. The fasciculus subcallosus of Muratow found in animals probably corresponds to the "substance grise sous-épendymaire" of Dejerine in man, in their opinion.

According to Obersteiner and Redlich a bundle, the size and thickness of the so-called fasciculus occipito-frontalis in a brain lacking the corpus callosum, does not exist in a normal brain.

Sachs (16) stated that a bundle of fibers corresponding to the " faisceau occipito-frontal " of Dejerine, extended vertically in the direction of the corona radiata lateral to the fasciculus nuclei caudati and above the caudate nucleus and that these fibers according to Wernicke came from the knee of the corpus callosum and entered the internal capsule.

Anton (17) described the brain of a three and a half year old boy in which the corpus callosum was absent in the anterior third and posteriorly consisted of a thin layer which did not unite in the median line. In this brain he found a fronto-occipital association system in the roof and the outer wall of the ventricle, which was situated medially from the corpus callosum and which sent fibers to the anterior limb of the internal capsule.

Tapetum.—The relation that the tapetum holds to the corpus callosum and the fasciculus occipito-frontalis also is a matter of dispute.

There are some authors who claim that the tapetum is in large part or entirely made up of fibers from the corpus callosum.

On the other hand, Onufrowicz, Kaufmann and Hochhaus believed that their cases prove that the tapetum is independent of the corpus callosum and that it really belongs to the fasciculus occipito-frontalis.

Muratow's experiments showed that the fasciculus subcallosus and the tapetum belong to one and the same system and he believed that little or no relation existed between the tapetum and the corpus callosum.

In this connection the experiments of Bianchi (21) upon apes have a distinct bearing, as he was not able to trace the degeneration of the fasciculus occipito-frontalis behind the parietal operculum. Dejerine believed that there is little or no relation between the tapetum and the corpus callosum, and Forel claimed that the fibers of the so-called tapetum did not belong to the corpus callosum but were association fibers of one hemisphere. He believed that that part of the corpus callosum tapetum going to the posterior horn belonged to the fasciculus occipito-frontalis of Forel.

The tapetum does not, according to Edinger (22), enter into the formation of the corpus callosum and he considered the tapetum, the posterior radiation of the fasciculus occipito-frontalis.

The case to be described below offered an unusual opportunity to study the relation which the tapetum holds to the fasciculus occipito-frontalis, inasmuch as there was present in the brain a hemorrhage which cut the tapetal fibers on one side, though to a somewhat limited extent. The other lesions in the brain were such as not to confuse the findings and permitted, I believe, definite conclusions to be drawn. The findings were so radically different in some respects, though confirmatory in others of the facts already described, that the conclusions are put forth conservatively and with some hesitation. The case has already been reported but not in this connection.

From the history of this case the hemorrhage evidently antedated death thirty-one months, thus giving ample time for secondary degeneration to become complete.

There was a hemorrhage in the outer and posterior wall of the descending horn of the lateral ventricle on the right side. The upper limit of this hemorrhage was observed as a fine line in the white matter of the posterior and external portions of the

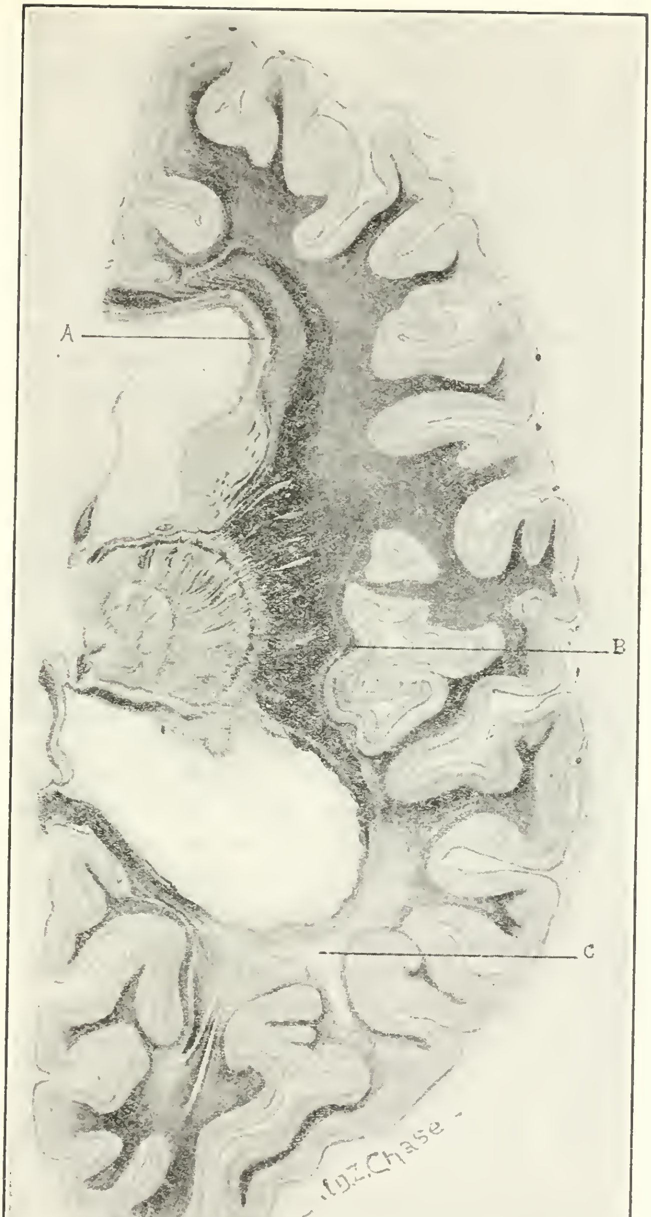

FIG. 3. (Right side.) *A*, degenerated fasciculus occipito-frontalis; *B*, degeneration in the posterior segment of the internal capsule secondary to the necrotic area in Fig. 1; *C*, hemorrhage involving the fasciculus longitudinalis inferioris and optic radiations of Gratiolet and tapetum.

occipital convolutions in the upper levels of the brain, corresponding to about the level where the caudate nucleus first makes its appearance in horizontal sections.

In sections just below this the hemorrhage was more extensive, involving the white matter of the posterior part of the corona radiata and extending forward to the posterior external angle of the distended lateral ventricle to within 3 mm. of the wall of the ventricle.

In the sections a little lower, the hemorrhage destroyed all the white matter between the tapetum and the gray matter of the first temporal and lower parietal convolutions, where it extended on each side of the inter-parietal fissure.

At the next level the optic thalamus appears in the section, the internal capsule is well formed, and the tail of the caudate nucleus is very small. Here the hemorrhage has involved all the white matter between the gray matter of the second parietal and first temporal convolutions and the descending horn of the lateral ventricle which is much distended (Fig. 3, *c*). The distance from the wall of the lateral ventricle to the cortex at this level is 2.5 cm. The white matter at this level measures 7 mm. in width, and is entirely destroyed by the hemorrhage, which extends from its anterior to its posterior limits a distance of 2.5 cm. It is at this level that the tapetum is implicated in the hemorrhage.

The lower limit of the hemorrhage is at a level where the red nucleus first begins to make its appearance. The lateral ventricle is very much distended and the white matter at the base of the third temporal convolution between the ventricle and the gray matter, measures one to three millimeters. This level corresponds to Fig. 231 in Dejerine, which passes 72 mm. below the superior border of the hemisphere.

While this lesion forms the basis for the anatomic study of the tapetum and fasciculus occipito-frontalis in this paper, mention should be made of a small area of softening (Fig. 1 *c*) which is observed in the white matter of the corona radiata in its middle portion, just internal to the fissure of Rolando on the right side. This necrotic area measures in its lateral diameter 1 cm., in its anterior posterior diameter 2 mm. and it extends in a vertical direction for about 3 mm. It is situated 3 cm. below the superior border of the right hemisphere about its middle portion.

In addition to this there is a third hemorrhagic area in the left optic thalamus. It is only the first hemorrhage, however, that has any bearing upon this study.

The entire brain was cut in serial sections. Of these, 28 on the right side and 15 on the left side were selected for description.

In section no. 1 (see Fig. 1) the level corresponds to Fig. 219 in Dejerine except that at this level the much distended lateral ventricle appears in the section. The occipito-frontal bundle stains less distinctly than normal and is evidently degenerated. This degeneration is continuous with a bundle of fibers which takes the stain less distinctly than normal, and which can be traced into the corpus callosum and around to the inner side of the ventricle, and thence backwards until it joins a band of degeneration which skirts the posterior part of the lateral ventricle, and which then extends forward in the external wall of the lateral ventricle to join the degenerated area anteriorly, in the position occupied by the "faisceau occipito-frontal" of Dejerine (Fig. 1, *A–B, E*).

The white matter at the base of the second parietal convolution stains very poorly and this is also true of the white matter of the second parietal convolution itself (Fig. 1, *D*). The fasciculus longitudinalis superioris appears to stain less deeply than normal as far forward as the white matter of the frontal lobe.

At this level to be emphasized then is the fact that more or less distinctly can be traced a band of degeneration surrounding the lateral ventricle and adjacent to its walls, and also the presence of degenerated white matter of the two parietal convolutions which is connected with a white line of degeneration situated at the base of the second parietal convolution just internal to the interparietal fissure, which appears in the midst of the fasciculus longitudinalis superioris and which can be traced along this bundle to the frontal lobe. The white matter here between the interparietal fissure and the wall of the ventricle measures less than a half a centimeter.

The fibers in the region of the fasciculus occipito-frontalis are not totally degenerated evidently as they take the stain to a certain extent.

A little lower than this level however, the band of degeneration surrounding the lateral ventricle can only be followed on the inside of the ventricle as far back as the posterior fifth, and on the outer side of the ventricle to the second fifth of the distended lateral ventricle. It is still marked however in the posterior wall of the ventricle.

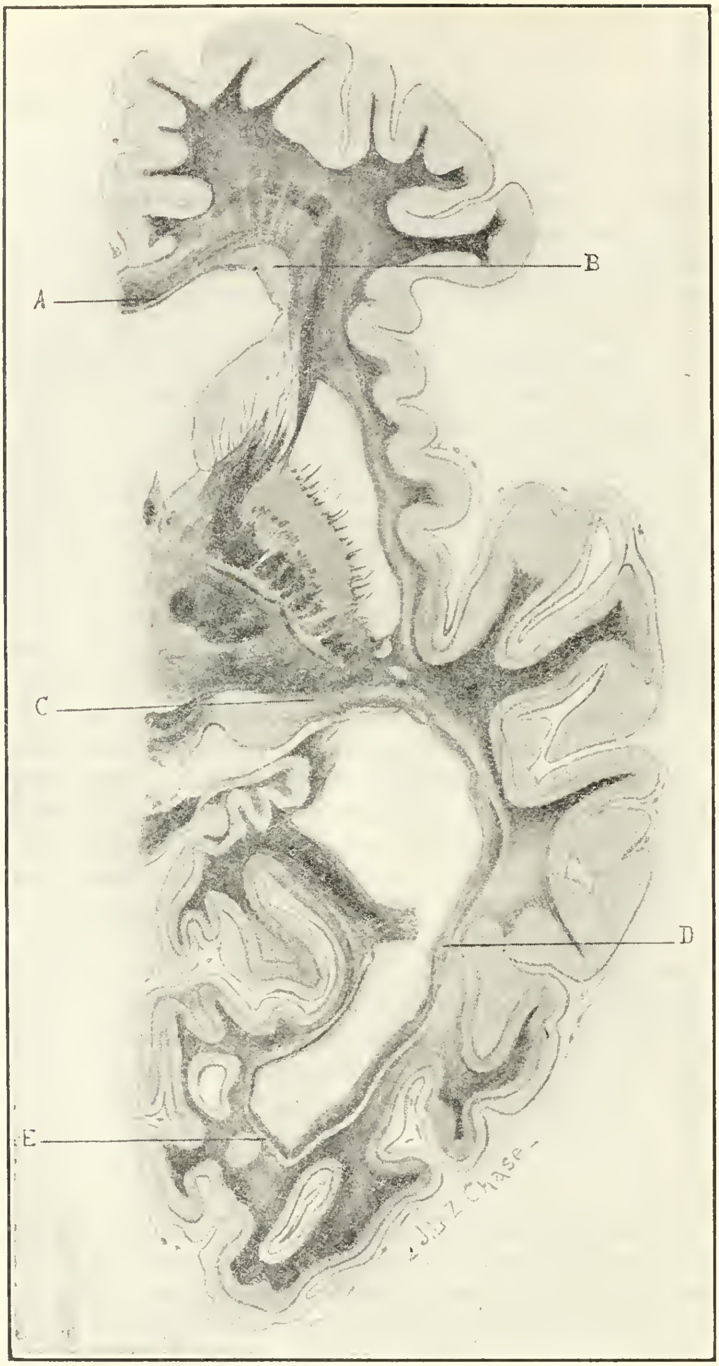

FIG. 4. (Right side.) *A*, degeneration of the corpus callosum; *B*, degeneration of the fasciculus occipito-frontalis; *C*, degeneration of fibers from the frontamen; *D*, lowest level of the hemorrhage; *E*, degenerated area continuous with the degenerated fasciculus longitudinalis inferioris.

In section no. 5 (see Fig. 2) the caudate nucleus just begins to appear in horizontal section. At this level the fronto-occipital fibers stain poorly and are continuous with a degenerated bundle which extends across the corpus callosum to the median line just forward of the anterior ventricular wall. There are between the wall of the ventricle and the area of degeneration, normally staining fibers which give the appearance of a dark narrow line in this region. Posteriorly there is a band of degeneration which can be traced from the border of the caudate nucleus backwards and around the lateral ventricle and forward again into the splenium. This is a fine line and there is between it and the wall of the ventricle a fine band of well-stained fibers.

The white matter of the occipital lobe is degenerated and this degeneration can be traced forward and outward to join the hemorrhagic softening above described as occupying the white matter at the base of the second parietal convolution.

The white matter of the first limbic lobe is also degenerated.

Section no. 6 (see Fig. 3) corresponds to Fig. 222 in Dejerine which passes 48 mm. below the superior border of the hemisphere. At this level the three nuclei of the optic thalamus make their appearance. The occipito-frontal fibers do not stain well and appear to be continuous with a band of degeneration which passes through the corpus callosum to the median line (Fig. 3, *A*). The external capsule stains poorly.

Just posterior to the tail of the caudate nucleus there extends for a distance of 2 cm. a band of degenerated fibers. It is at this level that the hemorrhage is most extensive and involves the entire white matter between the second parietal convolution and the wall of the ventricle at the posterior external angle of the ventricle, cutting at this level the fasciculus longitudinalis inferioris, the optic radiations of Gratiolet and the tapetum. Bands of degeneration are traced back into the occipital lobe and into the splenium from the necrotic area. Immediately forward of it, the white matter takes the stain fairly well with the exception of the small band of degeneration described above as extending posterior to the tail of the caudate nucleus.

In sections nos. 10 and 11 the degeneration can be traced in the white matter of the temporal lobe and the first occipital lobe. The fasciculus longitudinalis inferioris and the optic radiations of Gratiolet are both degenerated, but this cannot be traced as far forward as the internal nucleus of the optic thalamus. The tapetum is degenerated at this level. The white matter of the first temporal convolution and superior parietal convolution as well as the white matter just posterior to the posterior wall of the descending horn of the lateral ventricle is degenerated.

Beginning rather suddenly just beyond the hemorrhagic area, the fasciculus longitudinalis inferioris and the optic radiations of Gratiolet stain well and appear to be normal as far forward as the internal nucleus of the optic thalamus, but these bundles posterior to the hemorrhage are degenerated as far back as the posterior pole of the second occipital convolution. The internal capsule stains well except at one portion in the posterior segment of the internal capsule.

Section no. 14 corresponds to Fig. 224 in Dejerine which is 55 mm. below the superior border of the hemisphere. At this level two of the nuclei of the lenticular nucleus appear in the section.

Degeneration of the fasciculus longitudinalis inferioris and optic radiations of Gratiolet can be traced from the putamen backwards and outwards to join the area of degeneration at the base of the anterior portion of the two temporal convolutions. The tapetum at this level stains well. The white matter of the occipital lobe is not degenerated except for a band of degeneration at the posterior limit of the posterior horn which extends forward on either side of the posterior horn for a short distance, connecting on the external side of the ventricle with the hemorrhagic focus. The occipito-frontal fibers at this level are undoubtedly degenerated and appear still to be continuous with the degenerated band extending to the median line of the corpus callosum.

In section 16 there is a small necrotic area in the optic thalamus, just internal to the "zone réticulée" and nine millimeters in front of the posterior border of the optic thalamus.

In section no. 20 (see Fig. 4), Fig. 227 in Dejerine, the degeneration is still seen in the internal capsule faintly. The fasciculus occipito-frontalis stains better than in the levels above, though is evidently degenerated still. In the optic thalamus there is found a small almost microscopic hemorrhage in the internal medullary layer ("lame médullaire interne") which is evidently a continuation of the hemorrhage described in section no. 16.

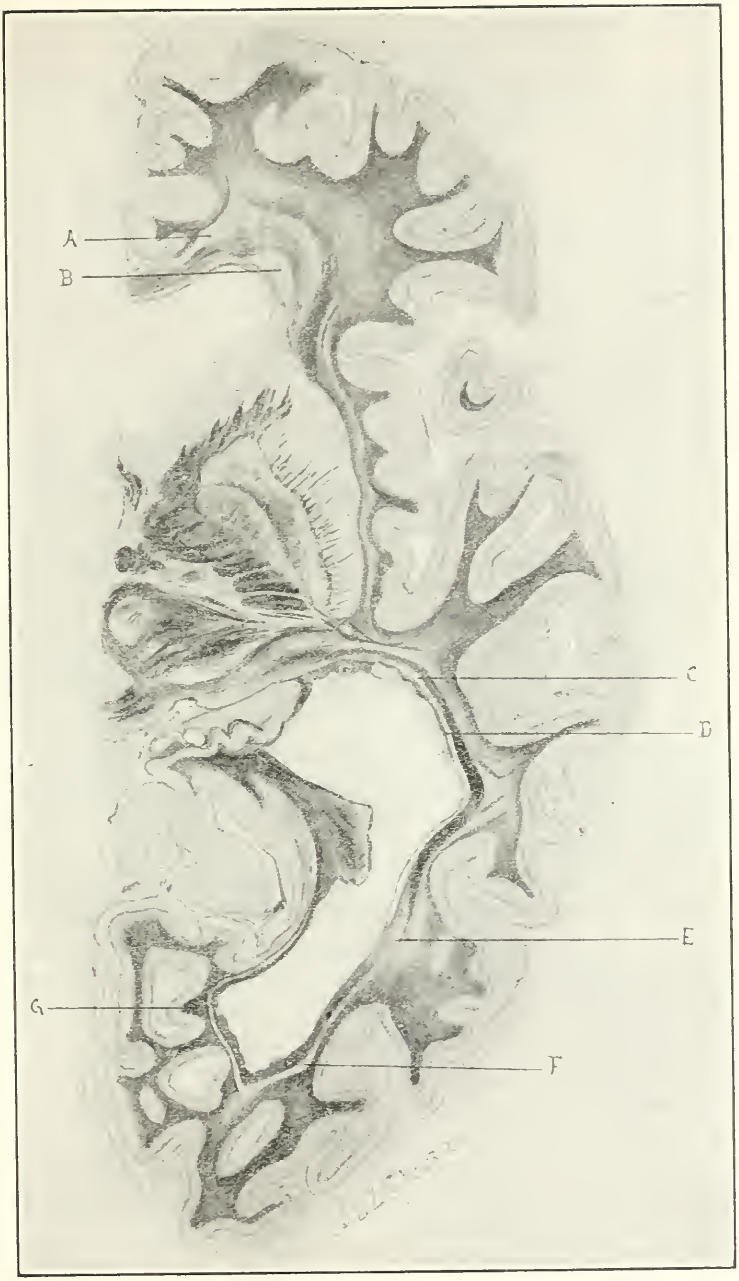

Fig. 5. (Right side.) *A*, degeneration of the white matter of the frontal lobe which can be traced into the corpus callosum; *B*, degenerated fasciculus occipito-frontalis; *C*, degenerated optic radiation of Gratiolet; *D*, tapetum which shows some degeneration; *E*, degeneration of the fasciculus longitudinalis inferioris and optic radiation of Gratiolet; *F* and *G*, degenerated area continuous with the degeneration found at *E*.

No degeneration could be traced from this area to the internal capsule.

At the posterior part of the first temporal convolution the optic radiations of Gratiolet and the fasciculus longitudinalis inferioris stain faintly for a space of about two centimeters. Anteriorly from this however, as far as the putamen, they stain very little. Posteriorly these bundles stain very poorly as far as the area of softening which, at this level is very small and at which point the distance between the gray matter of the cortex and the ventricle is but 2–3 mm. The white matter of the posterior part of the first temporal convolution and the anterior part of the second temporal convolution is much degenerated. From the area of softening posteriorly there is a band of unstained fibers which surround the posterior wall of the inferior horn of the lateral ventricle, and this can be traced forward into the forceps minor.

The white matter of the cuneus is slightly degenerated as is also the cuneo-limbic gyrus. The degeneration in the latter can be traced around the posterior wall of the lateral ventricle and forward to the point at which the calcarine fissure unites with the parieto-occipital fissure, where it joins the degenerated area in the forceps minor traceable to the fasciculus longitudinalis inferioris. The tapetum is small, stains faintly and is evidently somewhat degenerated.

At section no. 22 (see Fig. 5) corresponding to Fig. 229 in Dejerine, the internal and external geniculate bodies and the pulvinar appear to be smaller than on the left side. The fasciculus longitudinalis inferioris and the optic radiations stain fairly well as far back as the third occipital convolution, when quite suddenly they cease to stain, more especially the fasciculus longitudinalis inferioris. The tapetum is degenerated at this level, though posterior to the tail of the caudate nucleus it stains fairly well for a distance of about two centimeters.

The fasciculus occipito-frontalis does not stain well here, and degeneration can be traced forward to the median line in the knee of the corpus callosum.

The white matter of the third occipital convolution stains poorly, and the white matter of the cornu ammonis is degenerated. The degeneration in the fasciculus longitudinalis inferioris extends forward but cannot be traced into the cornu ammonis, but a little below this level it is possible to do so.

In section no. 25, the level corresponds to Fig. 230 in Dejerine, which is 70 mm. below the superior border of the hemisphere. The commissure of Meynert is intact as well as the internal geniculate body. Wernicke zone is apparently normal as is also the anterior commissure. The internal capsule shows a slight area of degeneration still. At the second occipital lobe the white matter is degenerated and also the white matter of the lingual lobe, which seems to be continuous with the degeneration in the occipital lobe. The fasciculus longitudinalis inferioris and the optic radiations stain very well as far back as the beginning of the second occipital lobe and suddenly then cease to stain. The tapetum is degenerated at this level, beginning about 1 cm. posteriorly to the caudate nucleus, forward of which the fibers take the stain fairly well though evidently the bulk of the tapetum is diminished.

In section no. 28 (see Fig. 6) the fasciculus longitudinalis inferioris stains fairly well up to the base of the second temporal convolution, then for a space of 1.5 cm. stains poorly. Forward posterior to the tail of the caudate nucleus the optic radiations are degenerated for a distance of 2 cm. Then a fine line of degeneration can be followed from this point backwards around the posterior horn and thence forward into the cornu ammonis. The tapetum at this level is almost entirely degenerated except for about a distance of 1.5 cm. posterior to the caudate nucleus. Just above this level the fasciculus longitudinalis inferioris and the optic radiations are almost completely degenerated.

In sections nos. 31 and 32, corresponding to Figs. 230 and 231 in Dejerine, there is some degeneration of the second temporal convolution. The fasciculus longitudinalis inferioris stains well at this level, and the optic radiations stain fairly well from the external geniculate body backwards to the beginning of the second occipital lobe. Here the staining suddenly becomes poor and a narrow band of degeneration can be followed posteriorly around the posterior horn and forward into the lingual lobe. The tapetum at this level is degenerated.

Left Side.—In the uppermost levels no pathological change can be observed.

In section no. 1 (see Fig. 7) a degenerated tract can be seen in the corpus callosum corresponding to a similar area of degeneration on the right side, and this can be traced into the fasciculus

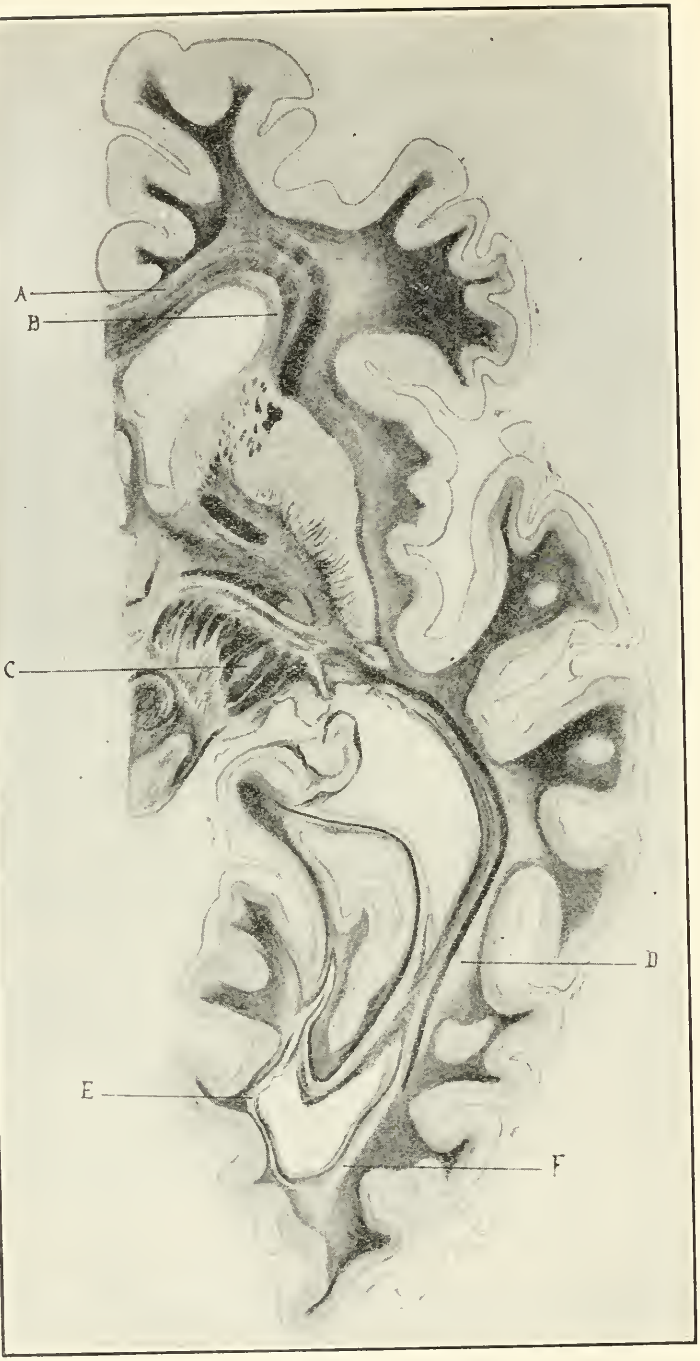

Fig. 6. (Right side.) *A*, degeneration in the corpus callosum arising apparently in the frontal lobes; *B*, degenerated fasciculus occipito-frontalis; *C*, secondary degeneration in the foot of the peduncle; *D*, degeneration in the white matter of the second temporal convolution; *E* and *F*, degenerated area continuous with the degeneration of the optic radiation of Gratiolet.

occipito-frontalis. Farther forward in the corpus callosum is a tract of degeneration which can be traced forward into the frontal lobe.

At a little lower level (see Fig. 8) the same condition is observed. Posteriorly here, however, in the corpus callosum there appears to be some degeneration extending from the opposite side to the tapetum. This degeneration cannot be traced at a lower level than that corresponding to Fig. 223 in Dejerine.

In section no. 6, the optic thalamus is the seat of an area of necrosis situated in about its middle portion. The fasciculus longitudinalis inferioris is slightly degenerated and the optic radiations of Gratiolet do not stain as well as normally in the posterior outer angle of the lateral ventricle.

In section no. 9, which is still lower and in which the three divisions of the lenticular nucleus, the red nucleus in the pons, and the anterior commissure, are seen corresponding to about Fig. 228 in Dejerine, the degeneration in the optic radiations of Gratiolet is more marked.

In section no. 15 the fasciculus occipito-frontalis is degenerated slightly, and this degeneration can be traced forward into the corpus callosum. The optic radiations of Gratiolet are somewhat degenerated at this level.

Résumé.—In the upper levels on the right side is a band of degenerated fibers more intense in the posterior wall of the lateral ventricle, which can be traced to the hemorrhage in the levels below which destroys the tapetum to a certain extent. This band of degenerated fibers is continuous with a faint line of unstained or poorly stained white matter which at the uppermost levels surrounds the lateral ventricle, leaving a band of well stained white matter between it and the ventricular wall. Forward and in the outer wall of the ventricle this band corresponds to the position of the fasciculus occipito-frontalis as described in Marburg's (23) atlas recently published (Fig. 71, *rcc*).

At a little lower level this band cannot be traced all around the ventricle but is present still to a marked degree in the posterior wall of the lateral ventricle.

These areas of degeneration, I believe, may be probably interpreted as being fibers of the fasciculus occipito-frontalis which have been pushed to the side by the distension of the ventricle.

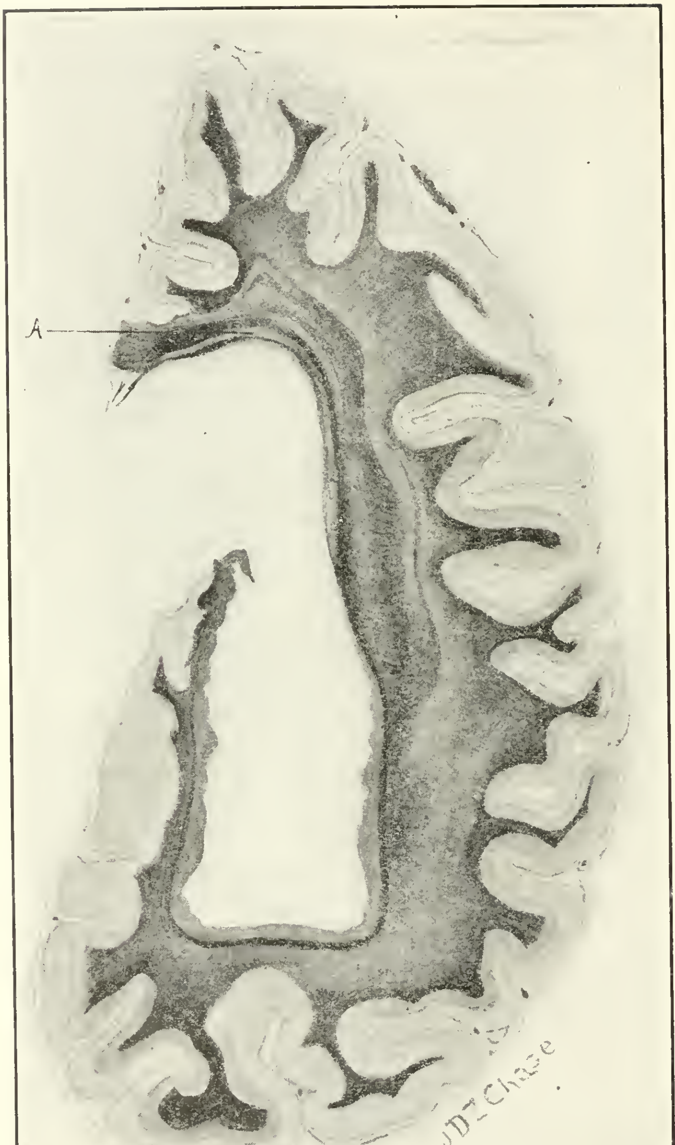

FIG. 7. (Left side.) *A*, degeneration in the corpus callosum.

At levels still lower the fasciculus occipito-frontalis is undoubtedly degenerated, and the unstained bundle of fibers can be traced into the corpus callosum as far as the median line, and when the same level on the left is studied this can be followed into the fasciculus occipito-frontalis on this side.

What appears to be degeneration can be traced into the splenium on the right side, but what the fate of these fibers on the opposite side is cannot be determined. Probably though, they extend into the tapetum of the left side, as in one section the tapetum was degenerated and degenerated fibers could be seen in the splenium.

There is little doubt that the degeneration of the fasciculus occipito-frontalis can be directly traced to the degenerated tapetum on the right side. That the degeneration in the corpus callosum is continuous with the degeneration in the fasciculus occipito-frontalis seems more than probable. This finding is uniform in a great many sections, and in comparison with sections from another brain recently studied, it appears to be undoubtedly abnormal.

These findings support the views of those who claim that the tapetum is a part of a long association bundle connecting the frontal and occipital lobes. Whether the tapetum is made up exclusively of fibers from the fasciculus occipito-frontalis or in part from the corpus callosum cannot be definitely stated from the findings in this case, in spite of the fact, that degenerated fibers could be found in the splenium directly traceable to the original hemorrhagic focus, since the white matter of the occipital and temporal lobes was implicated as well.

In conclusion, it seems probable that there is a long association bundle corresponding in position to the "faisceau occipito-frontal" described by Dejerine, of which the tapetum is a part, and which sends some fibers across the knee of the corpus callosum to the fasciculus occipito-frontalis of the opposite side.

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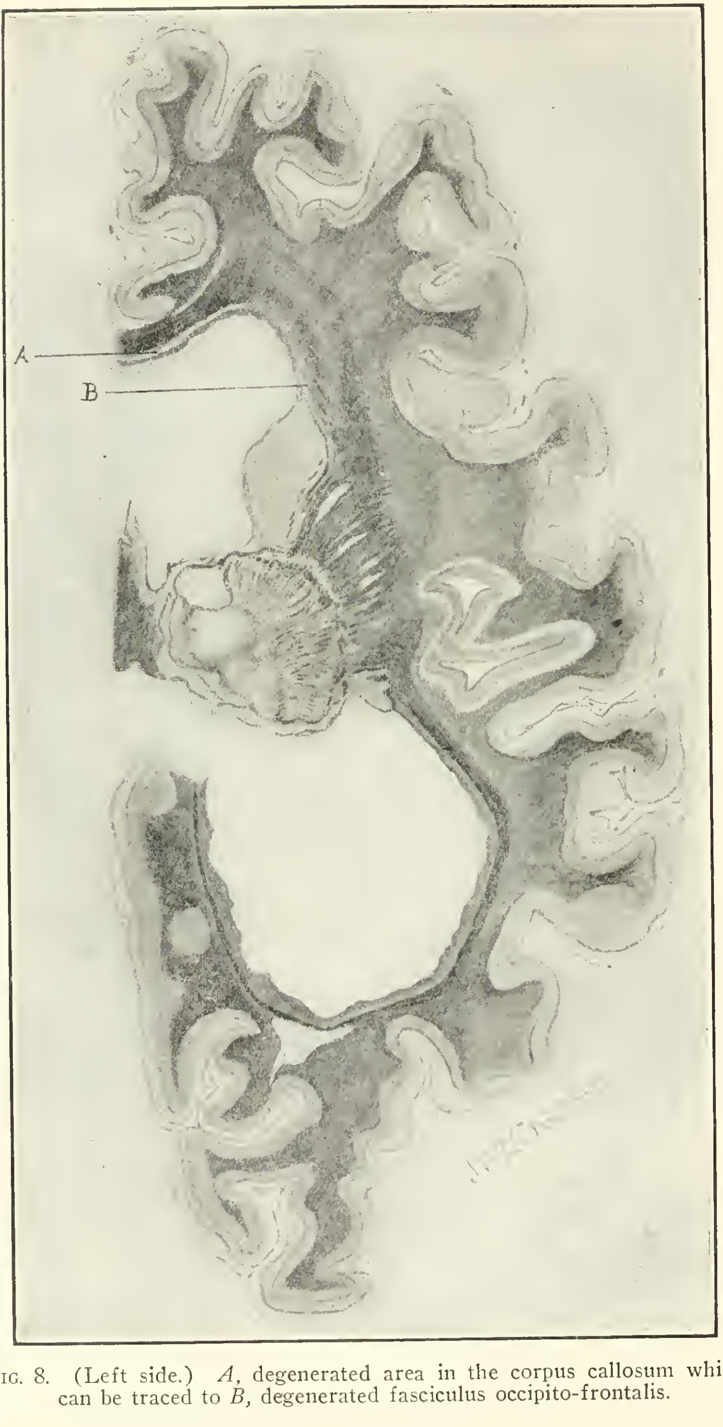

Fig. 8. (Left side.) *A*, degenerated area in the corpus callosum which can be traced to *B*, degenerated fasciculus occipito-frontalis.

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COLLOID BODIES IN THE CENTRAL NERVOUS SYSTEM: THEIR PRESENCE AFTER SEVERE TRAUMATISM IN A CASE OF DEMENTIA PARETICA[1]

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It appears that the first knowledge we have of the recognition of so-called colloid bodies dates back to the observations of Zenker,[1] an ophthalmologist, who described what seemed to be varicosities on the axis cylinders in the retina. His observations were confirmed later by Müller,[2] and others have described the occurrence of these bodies in the retina after traumatism. A few years later, Tuke and Rutherford[3] observed what they believed to be a new lesion in the brain of a subject who had died after two attacks of apoplexy. In the white matter of the atrophied areas they found semi-opaque whitish spots which in thin sections could be seen with the naked eye. They were almost entirely confined to the white substance, and when magnified, were seen to consist of what they termed molecular matter, and they believed the diseased elements were the nuclei of the neuroglia. It was there that the term miliary sclerosis was first employed, a designation of these bodies which has since passed out of use.

These observers laid stress upon the fact that the lesions found by them differed from those described by Rindfleisch.

Virchow[4] found similar bodies in a brain undergoing softening from encephalitis. Later, Tuke[5] considered the subject of colloid degeneration under the heading of disease of the neuroglia, while Lewis,[6] not disputing the origin of this substance, stated that he found bodies which could not be distinguished from those described by Tuke, which were not caused by sclerosis or degeneration of the connective tissue elements, and which were without doubt the direct product of morbid action in the medullated nerve fibers. It was he then who first pointed out this origin. These bodies he described as being spheroid, homogeneous, clear, pellucid and colorless, devoid of crescentic markings, frequently having crenulated margins, and varying in size from 13μ to 27μ, and even as much as 40μ in some positions. They were not affected by iodin and sulphuric acid, resisted carmine, but stained a light purplish blue with hematoxylin. His reasons for believing they originated from the medullated fibers were: their variations in diameter bore a direct relation to the diameter of the nerve fibers where they were found; they were limited to the regions of the larger nerve fibers; there was no structural analogy between these bodies and the elements of the neuroglia, and a direct communication with the medullated nerve fibers was detected. At that time he made a noticeable difference between the bodies and miliary sclerosis. The latter lesion he then asserted was quite common in brains of the chronic insane, but some years later he wrote[7] that the colloid body was attached, forming an integral part of the medullated fiber and hence not removable like the miliary products. He regarded both as representing stages in the progress of a chronic degeneration of the medullated fibers. Kesteven[8] observed that the transition from the "colloid" to the "miliary" formation had occurred in one of his cases, an observation which Lewis later accepted as conclusive. Under the heading of colloid degeneration Lewis[9] describes these bodies as identical in their nature with colloid bodies, and further states that the name is unfortunate since it assumes a colloid transformation of a connective tissue cell similar to what occurs in the epithelium of the thyroid gland.

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[1] Read before the Philadelphia Neurological Society, October 28, 1910.

It is interesting to note that Tuke[10] later agreed with Lewis by his statement, "Miliary sclerosis is not a primary sclerotic change but is an accident occurring in the course of a subacute inflammatory process."

The first experimental work bearing upon this matter of which I have been able to find record was that of Tuke,[11] who confirmed the work of M'Kendrick, and found by inflicting injuries upon the heads of pigeons, small colloid bodies in every instance where the case was of ten days' standing. Hamilton,[12] by passing threads through the spinal cords of animals, was the first to produce an artificial myelitis; at the end of forty-eight hours he examined the cord and found these bodies present, but he believed they were derived from the axis cylinders. Woodhead[13] observed similar bodies in a case of locomotor ataxia. In this connection Tuke, in writing of the amyloid bodies of Virchow, remarks: "Contrary to the observations of many, I have invariably failed to obtain any reaction by the application of iodin, nor can I feel certain of the existence of concentric rings." The difficulty of identifying bodies of this nature may be appreciated when we bear in mind the italicized words of Wells:[14] "*The word colloid is merely morphologically and macroscopically descriptive: there is no definite substance colloid.*"

Spiller[15] has reported his findings in a case of amyotrophic lateral sclerosis with bulbar symptoms, and believes that the bodies there seen resemble more closely the colloid bodies described by Lewis than any others with which he is familiar. The noteworthy observations of Miles[16] have shed considerable light upon this subject. His studies were made from a large number of cases where death had resulted from severe cerebral traumatism; one had been a case of syphilitic encephalitis; he also experimented extensively upon birds, pigs and rabbits. These bodies were found in one bird which had survived its injuries only a few minutes, thus convincing him that disease was not necessary for their production, though it was by far the most common cause. He believed when they were not found in the white matter that they had been carried forward by the blood or lymph current. When due to traumatism, he accounted for their production by applying Duret's cerebro-spinal fluid theory of the mechanism of cerebral concussion—that the "cone" of depression resulting from the blow causes compression of the nerve fibers through increased tension, which is followed by the escape of myelin through the slits of Lantermann or by a bursting of the sheath. This is not at all improbable when we remember that myelin is generally described as a fine emulsion contained within the meshes of a delicate reticular substance, and from the broken ends of a fiber irregular droplets are extruded. Myelin very soon undergoes post-mortem changes.

Woodhead speaks of these substances in the edition[17] of his work which is just out; he believes the bodies result from inflammation and may be due to injury or disease, and he states that they are apparently droplets of myelin set free from the inflamed or degenerating white substance of Schwann, or in some cases it may be from the degenerating axis cylinders.

Most observers of this condition have been accused of studying artifacts, a charge which has been abundantly disproven by the repeated finding of the same bodies in frozen sections taken from fresh brains. In the light of our present knowledge it seems that the so-called colloid bodies may result from traumatism or from an inflammatory condition, and it appears that the origin of these bodies is from the myelin of the medullated nerve fibers, and rarely, perhaps, from fragmentation of the axis cylinder.

The condition of colloid degeneration seems to belong to another subject. It has been carefully studied by Alzheimer[18] and others. Here the various vessels are chiefly implicated, sometimes to the extent of ensheathing them.

The following report is of a case in which colloid bodies were found in great profusion.

The patient, a colored man, aged 32, had become sufficiently insane to call for institutional detention for the nine months preceding his death. The clinical history during his detention was so clear as to admit of no diagnosis except that of dementia paretica. He died twenty hours after having sustained repeated and severe blows to the head. At a necropsy made a few hours after death, the cerebellum was observed to be unusually small (probably congenitally so). There was no evidence of hemorrhage either upon the cortex or within the substance of the brain. The usual gross appearances of chronic meningo-encephalitis was present. The brain was placed in formalin and later passed through alcohol.

Upon microscopic examination, the bodies were observed in many parts of the brain and were found as far down as the pons. They were not detected in the cerebellum and medulla. Not more than half an inch of the cord was available, and in this they were absent. The part showing the greatest abundance was the optic chiasm; and in this connection it is interesting to note that the left eye had been dislocated, an occurrence which must have caused considerable traction upon the optic chiasm, and in a brain which had for many months been subject to a chronic meningo-encephalitis, it does not seem at all improbable that the myelin sheaths were ruptured with extrusion of particles of myelin. The diameter of the bodies for the most part fell within the range of 15μ to 25μ; a few were smaller while some were well beyond this size. When viewed through a Nicol prism, they showed no polarization. The reaction with osmic acid was uncertain, and I was not able at any time to trace a direct connection between the particles and the nerve fibers; they did not stain with iodin, nor were they affected by sulphuric acid; a pinkish hue was obtained with hematoxylin. The most intense staining quality was exhibited when toluidin blue or thionin was used. The former gave a deep purple color, while the latter imparted a distinct amethyst tinge to this substance. After a few days the color began to fade, and by the end of three weeks it had entirely disappeared from the bodies. Three months later areas were again studied, the brain having been preserved in formalin, with but little loss in the staining property.

Photographs fail to adequately show these bodies. They are not nearly so distinct as when viewed through the microscope, since in the latter instance their different standing quality causes them to stand out in contrast with the surrounding tissue.

It would seem that these bodies might have medico-legal value if found after death in cases suspected of cerebral traumatism.

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EPILEPSY AND ITS MODERN TREATMENT

BY CHARLES K. MILLS, M. D., PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA

The disease epilepsy is one of the most ancient of which we have records, and these records are found not only in the earliest medical writings, but also in the pages of the poet and the historian. Hippocrates, who lived nearly five hundred years before Christ, wrote learnedly of epilepsy, and the poet Lucretius, who flourished in the century preceding the Christian era, gave in his *De Rerum Natura* a description of the epileptic attack which has not often been excelled in modern descriptive definitions.

It is not necessary for me to recall to this audience, many of whom are familiar with the literature of epilepsy, the names which were applied to the disease in early times. It may be worth while however to allude to one or two of them, and to discuss their appropriateness. One of these was *morbus sacer*, or the sacred disease, which term is said to have been used because the priests or priestesses before giving out their oracles fell in an epileptic fit, or what was supposed to be one. With our present knowledge of the disease and its counterfeits it is probable that these priestly seizures could be relegated rather to the domain of simulated epilepsy or to hystero-epilepsy. Skilful simulation of epilepsy is not very uncommon in the experience of those who deal with nervous diseases either from the standpoint of the clinician or of the medical jurist, and genuine hystero-epileptic seizures which, properly regarded, belong under grave hysteria, are not unlikely to occur in those who are in the habit of strongly appealing to their emotional natures.

The simulation of epilepsy was no more impossible to an ancient oracle or priest than to the modern faker. I have seen the disease so cleverly simulated by a man who was in the habit of counterfeiting not only this disease but others almost as difficult, that the deception would have easily misled anyone who had not foreknowledge or was not capable of applying the most expert methods of uncovering the deception.

The story is told of a clinical teacher, who while walking the wards with his students, discoursing on epilepsy, pronounced the opinion that the disease could not be simulated in a manner which he would not readily discover. Soon after a young physician in the company suddenly fell with a cry in a severe fit, frothing at the mouth and exhibiting some of the most striking phenomena of the epileptic attack. The clinician after an examination said that by a strange chance the company had before them an example of the genuine disease, whereupon the young man sprang to his feet declaring that he could cure his epilepsy as easily as he had caused it.

Some years since there was in Philadelphia a youth who was a victim of genuine epilepsy. At the time and after his seizures he was often surrounded by a crowd, who, moved by sympathy as he came to his senses, contributed somewhat freely of their small change. Finding that the business of having real fits was becoming dull, and having seen other epileptic attacks in the hospitals where he sometimes sojourned and also having heard them described, he took up the business of shamming epilepsy for the purpose of adding to his resources, which business he pursued with some success. Both the real and the counterfeit disease were recognized by some of those who investigated his case.

It might be interesting to an audience like this to know that another of the ancient names of epilepsy was *morbus commitialis*, or the disease of assemblages, so called because when an epileptic fit occurred in the midst of an assemblage all other business was for the time suspended, a precedence which it still holds in our own time in the same connection. For the ancients, however, its domination of the occasion was due to the superstitious awe with which epilepsy was regarded. In our own day it springs both from sympathy and from a desire to help the victim of the distressing disorder.

A word or two might be said regarding some of the great historic characters who are said to have been the victims of epilepsy. These include Julius Cæsar, Mohammed, Paul I and Peter III of Russia, and Napoleon. The evidence is not convincing with regard to all of them, but the subject is worthy of passing notice.

Suetonius* says of Cæsar, "he was tall, of a fair complexion, round limbed, rather full faced, with eyes black and piercing: and that he enjoyed excellent health, except towards the close of his life, when he was subject to sudden fainting fits, and disturbance in his sleep. He was likewise twice seized with the falling sickness while engaged in military service."

Plutarch† says of Cæsar, "he was a spare man, had a soft and white skin, was distempered in the head, and subject to an epilepsy which it is said first seized him at Corduba."

It must be remembered that in the times of Suetonius and Plutarch little was known of the effect of such diseases as cerebral tumor, brain syphilis, and toxemias of various sort causing convulsive attacks. That the distemper or disease from which Cæsar suffered came on late in life rather argues against the probibility of his having had what is known generally as idiopathic epilepsy, as do also his retention of unusual mental and physical powers and the excesses to which he was known to be addicted.

Gibbon‡ in the fiftieth chapter of his "Decline and Fall of the Roman Empire," says regarding the alleged epilepsy of Mahomet, "till the age of sixty-three years, the strength of Mahomet was equal to the temporal and spiritual fatigues of his mission. His epileptic fits, an absurd calumny of the Greeks, would be an object of pity rather than abhorrence; but he seriously believed that he was poisoned at Chaibar by the revenge of a Jewish female." In a note to this chapter of Gibbon, added by Dean Milman and Dr. Wm. Smith, however, these commentators say that the opinion of modern oriental scholars rather tends the other way. "Dr. Sprenger (p. 77) shows, on the authority of Ibn Isaac, that Mahomet, whilst still an infant under the care of his foster mother, had an attack which at all events very much resembled epilepsy. Three other fits are recorded (Ib. p. 78, note 4). Dr. Wiel (Mohammed, p.

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*Suetonius Tranquillus, C., translation of Alexander Thomson, M. D., revised and corrected, page 38, New York, 1883.

†Plutarch's Lives, the translation called Dryden's, corrected and revised by A. H. Clough, Vol. IV., page 273, Boston, 1859.

‡Gibbon, Edward, Life of Mahomet, fiftieth chapter of the Decline and Fall of the Roman Empire, with notes by Dean Milman and Dr. Wm. Smith, pages 169-170, New York, 1895.

26, note 11) remarks that the word Ussiba, which Abulfeda uses with regard to Mahomet, is particularly used of epileptic attacks. The same author has collected several instances of these fits, (Ib. p. 42, note 48, and in the Journal Asiatique, Juillet, 1842), and is of the opinion that his visions were, for the most part, connected with them.—S.''

Talleyrand* in his memoirs speaks of the epilepsy of the Russian Czar Paul I, who suffered from epilepsy as did his father, Peter III.

According to Dr. James Hendrie Lloyd,† belief in the epilepsy of Napoleon is founded upon a statement in the memoirs of Talleyrand, but he adds that this statement is not conclusive.

From the time of Hippocrates to the present, both masters and apprentices in medicine have continued to direct their attention to epilepsy. Writers on general medicine whose names are cherished in medical history have used their powers in the illustration of the phenomena, in the discussion of the nature and cause, and in the consideration of the best methods of dealing with the disease epilepsy. The results perhaps have not been commensurate with the time and labor bestowed.

Some time ago, when preparing an address on the subject of ancient and modern organo- therapy, I was especially interested in seeing how frequently epilpesy under its various designations was refered to in the books and articles which I consulted. Many times reference was made to this disease, and many forms of organic treatment were recommended for it, these usually being of such a fantastic, absurd, not to say nauseous character as only to create amusement or disgust in the mind of the modern reader. In recent times a revival of interest in the organic treatment of epilepsy has taken place, as in that of many other diseases; and yet I am bound to say that no results, worthy of much approval, have come from investigations in this direction. I refer to what has been attempted from the employment of cerebrin, for instance, or from the use (following some of the

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*Memoirs of the Prince de Talleyrand, edited by the Duc de Broglie, translated by Raphael Ledos de Beaufort, F. R. H. S., Vol. I, page 199, London, 1891.

†Lloyd, James Hendrie, article on Insanity, Wharton and Stille's Medical Jurisprudence, fifth edition, pages 733-734, New York, 1905.

Italian physicians) of the serums, from the blood of epileptics; or of thyroid gland, or of thyroidectine, made from the blood of animals from whom the thyroid has been removed. At one time it looked as if something hopeful might be obtained in this direction, as in other diseases, from the use of these organic preparations.

Universal interest has always been taken in this disease, both by the medical profession and by the community at large; and this because of its wide dissemination, its dramatic attributes, the seriousness of its effects and the difficulties which surround its cure. Next to insanity and tuberculosis, epilepsy has an abiding interest for all who look to state medicine, as well as to scientific research, for the final solution of the problems of health and disease. It is for this reason among others, that this State Conference of Charities and Correction has properly placed the subject of epilepsy upon its program.

Let it be understood just what the disease is of which I am talking. The term epilepsy is used somewhat loosely, its application being extended to include almost any form of structural or toxic disease having spasms or convulsions among its manifestations. Doubtless many of the cases spoken of in ancient medical annals as epilepsy, the falling sickness, the sacred disease, and by many other designations, did not belong in this category according to the modern acceptation of the word. One of the important results of the investigations of the last half century has been the separation of a considerable number of convulsive diseases with traceable causes, from epilepsy, properly so-called. Tumors, cysts, abscesses, hemorrhages, meningitis, toxic affections, like uremia, diabetes, and alcoholism, are now known not only to have spasmodic attacks as a part of their clinical history in many cases, but also the diagnostic methods of determining the nature of these affections have been thoroughly studied. Hystero-epilepsy, which belongs in the domain of grave hysteria, has also received its proper place in medicine. Physiological experimentation, hospital and institutional observation and pathological investigation have all come to our aid in isolating epilepsy from diseases with which it has some points in common. In hospitals or institutions for epileptics instances of any of the above forms of disease may often be found, but the epilepsy with which we are chiefly concerned is a chronic, progressive disease which has at its basis some constitutional condition, usually associated with a neuropathic inheritance. Let me turn for a few minutes to a consideration of the frequency of this disease, as this is a matter of special interest to this audience.

Returns for the whole country are not sufficiently available to enable us to give anything like an accurate statement with regard to the whole number of epileptics in the United States. It is probable, however, that it would be under-, rather than over-estimating to put this number at fifty thousand. Of this number only a fraction are cared for by the state and by counties and municipalities. The majority are living, sometimes cared for and well treated, but often in absolute or relative neglect, at their own homes. Not a few of them are married and are adding to our defective classes.

In 1890, according to Diller*, there were in the various state insane asylums, almshouses, etc., throughout Pennsylvania under the jurisdiction of the State Committee on Lunacy, 615 epileptics the great majority of whom were indigent. These, this writer calculated, together with those in general hospitals, would then have probably footed up a total in institutions of over 1000. Diller estimated that there were at that time probably 4000 or 5000 epileptics of all grades living in Pennsylvania.

Twenty years ago there was not a single general institution in the United States supported by the public, municipal, county, or state, set aside for the care of epileptics and the study of epilepsy. Dr. Lucius Baker had at this time a small, semi-private institution for epileptics located at Baldwinsville, Massachusetts, with perhaps less than a score of patients. Much good was done by the establishment of this small hospital of Dr. Baker, not only for those committed to his charge, but also by its example stimulating public interest in the question of institutional care for these unfortunates. It was about this time and a little later that the subject of state provision for epileptics began to be seriously agitated in this country. Since that time a few of our states have made large, if not sufficient provision for the care of their epileptics, notably Massachusetts, Ohio, New York, New Jersey, Indiana, Maryland, and Kansas.

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*Diller, T., The Medical News, August 20, 1892.

In Pennsylvania the matter of the care of epileptics has in recent years not been neglected. Dr. Wharton Sinkler, Dr. Herbert Norris, and the speaker, with others, founded in 1898 an epileptic hospital in Philadelphia, first in connection with St. Clement's Episcopal Church. Soon this institution was expanded and received a number of munificent gifts from Mr. Henry C. Lea, Miss Rebecca Coxe, Mr. Eckley B. Coxe, Mrs. Travis Cochran, and others. The institution soon was removed and under a new name and with wider purposes was established as the Epileptic Hospital and Colony Farm at Oakbourne, Chester County, Pennsylvania, where it is now doing a work of great value for those who suffer from epilepsy, for their families, and for the community. This institution however is far from being sufficient to supply the demands made upon it, and always has a long waiting list of those desiring admission. In its main building and its cottages it can accommodate fewer than one hundred patients. Its grounds, while well located and admirably adapted to its purposes, are limited. In addition to private liberality, on which it has chiefly relied, it has received some aid from the state, particularly in the way of maintenance. On the whole, however, it can be said to care for less than a fortieth or fiftieth of those who need institutional care in this state. Its acreage should be trebled or quadrupled, and its accommodations for housing should be equally increased.

Just here while referring to the Pennsylvania Epileptic Hospital and Colony Farm at Oakbourne, let me step aside for a moment to pay a word of tribute to Dr. Wharton Sinkler, who has recently passed from among us, and who did as much as any single man in our commonwealth to advance the interests of the epileptic and to stimulate the study of epilepsy from the standpoint of science and of state medicine. His interest in the subject of epilepsy showed itself not only in valuable contributions relating to this disease, but also in his active efforts in connection with special institutions. In 1903 he was president of the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics. The inception of the institution, now at Oakbourne, was due to him. I had constant opportunity to observe the noble work which he did in connection with this charity. First as one of its physicians, later as one of its trustees and president of its board of management, he from the beginning successfully exerted himself in its behalf. Most of the money which was secured for the purchase of the property, erection of buildings, and endowment for maintenance, was obtained through his energy and because of the great respect and regard in which he was held by those contributing. An endowment of fifty thousand dollars as a memorial to him has recently been secured for this hospital.

A quotation from an article by Dr. Sinkler,* published in 1901, is of value as giving a resume of what had been attempted up to that time by Pennsylvania for its epileptics, and also as showing the number of epileptics in Pennsylvania at several periods from 1893 to 1901.

"The state has recently established a magnificent institution for feeble-minded persons at Polk, the capacity of which is 650, and of these 118 are epileptic. The question of caring for this unfortunate class by the state has been under consideration for several years, but so far little has been accomplished in providing for them.

"In 1893 the Board of Public Charities of this State made a strong appeal to the Legislature to establish an institution exclusively for epileptics. Again in 1895 the Board brought the matter to the attention of the Legislature, and in 1897 a bill was introduced into the Legislature, prepared by the Lunacy Committee, for the establishment of a State Hospital for Epileptics, but unfortunately this bill failed to pass. The need of separate institutions for epileptics is far greater to-day than it was in 1893, for the number of cases of epilepsy is increasing with the growth of the population. In 1893 the Board of State Charities recorded 575 cases of epilepsy in the different state institutions. The same board reports that, on September 30, 1900, there were 827 cases in the different state hospitals, asylums, almshouses and private institutions. This increase, which is apparently out of proportion to the growth of the population, is probably due to the fact that more epileptics have been received into state institutions, some of which, like the hospital at Polk and the Asylum for the Chronic Insane at Wernersville, have been opened since 1893. A personal canvass by means of letters which I have sent to the different state and private institutions in Pennsylvania, gives a still greater number of cases, and these figures are by no means complete, as the time at my disposal for the inquiry has been limited, and I have been unable to get replies from all of the institutions in the state. Many have failed to answer my letter of inquiry, as the time has been rather short since it was sent, and some of the smaller institutions and private sanitoriums I have been so far unable to reach. The replies which have been received, however, give the total number of epileptics in state and private institutions as 1164, which are distributed as follows:

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*Sinkler, Wharton, Condition of Epileptics in Pennsylvania, American Medicine, September 14, 1901.

" In state asylums and institutions for feeble-minded -	854
In general hospitals and private sanitoriums -	177
In county almhouses - - - - - - - -	116
In jails - - - - - - - - - -	11
In homes for children and aged persons - - -	6
	1164 "

Sinkler, reasoning on the imperfect returns received and on the basis that the epileptics outside of institutions were probably twice as many as those in them at the time of his investigation, concluded that we probably had one epileptic for about every 2000 of the whole population, or a little more than 3000 in the state. The actual number was probably in excess of this.

Since 1901 the increase in the number of epileptics has no doubt kept pace with the population. I have not made any elaborate effort to get the entire number of epileptics at present in institutions of all sorts in the state. Some data received in reply to inquiries, however, indicate a considerable increase since 1901. I believe that the epileptic population of Pennsylvania can be safely estimated as at least 5000 or 6000, and proper provision should be made by the commonwealth for at least 3000 if we hope to keep pace with the work being done in other states.

Through the courtesy of Dr. Frank Woodbury, Secretary of the Committee on Lunacy of the State Board of Charities, I have received the following data with regard to epileptics in institutions for the insane under the care of this committee.

"The report for the year ending September 30, 1909, is in the hands of the public printer. It gives for that period, males 656, females 549, total 1205. The reports from hospitals for the corresponding period of 1910 have not yet been sent to this office, but I hope to have them before the end of the month. I expect an increase of from 8 to 10 per cent. These figures do not include the epileptics at Oakbourne, Elwyn, Polk, or Spring City. I have no complete statistics for 1910 of the number in almshouses, but in 1909 there were 32; but this figure includes only the epileptics who have been certified as insane."

In the Eastern Pennsylvania State Institution for the Feeble-minded and Epileptic at Spring City, Montgomery County, Pennsylvania, according to a letter which I have recently received from Dr. H. M. Carey, the superintendent, the number of epileptics is 127, of whom 12 are primarily feeble-minded, and 115 are epileptic who are not strictly to be classed as feeble-minded.

At Oakbourne the number of patients is between 80 and 90.

During the present month an enumeration of epileptics in the Philadelphia General Hospital shows that besides 168 in the Insane department, there are 58 in the hospital and 22 in the out-wards.

In private sanitariums and several private houses where such cases are cared for, the number of epileptics in this state would probably reach about one hundred to one hundred and fifty.

These figures are far from indicating the number of epileptics under some form of institutional care.

In the study of disease many things may appeal to the scientific physician: its history, its similarity to other diseases, its diagnosis, and the theories of its nature and of its causation. That which most appeals to the lay mind is prevention, or treatment with the hope of cure. The questions which most readily fix general attention are, can epilepsy be cured, and if so in what percentage. Secondly, if it can be cured in a certain percentage, is this result usually or often obtained. The answer to the first of these questions is that even under present conditions, epilepsy, in perhaps ten per cent of the cases treated can be cured. When the question is asked, is epilepsy cured in this percentage, the answer must be that this is not the case. While this is true, I believe that the cures in epilepsy under most favorable circumstances could be made to reach at least as high as fifteen or twenty per cent.

With regard to the curability of epilepsy Dr. William Aldren Turner,[*] one of the ablest of recent writers on this subject, speaks as follows:

"Everyone who has had much practical experience of epilepsy knows how unsatisfactory the treatment of this disease is in a large number of cases, more especially in the later stages of the disease, when the bromides are of little or no avail. Moreover most methods and systems of treatment are of some benefit for a time; but the progressive character of the disease and the tendency towards mental deterioration, in the course of years assert themselves, and the patient passes into the class of confirmed epileptics for whom little or nothing can be done.

"There is, on the other hand, quite a considerable percentage of cases, varying from 25 to 50 per cent, in which a favorable outcome may be expected, as a result of carefully considered and well sustained treatment over a number of years. The following features, singly or in combination, may therefore be regarded as constituting a *favorable type* of the disease:

"(a) Cases in which the disease commences between the ages of 16 and 20 years; for the statistics show that as many of these have the disease arrested as become confirmed epileptics.

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[*] Turner, William Aldren, Epilepsy, A Study of the Idiopathic Disease, 1907.

"(b) Cases in which the disease commences after the age of 45 years: in other words, the so called 'late epilepsy'.

"(c) Cases in which seizures are not more frequent than once or twice a year. This is an especially favorable feature, and its existence should prompt to continuous bromide medication for a number of years.

"(d) The absence of any obvious mental impairment.

"(e) The absence of numerous or pronounced structural stigmata of degeneration.

"In contrast to the above, there are certain features which condemn the patient almost from the outset to incurability. Amongst the chief *unfavorable features* may be mentioned:

"An early commencement of the disease, more particularly when under five years of age; great frequency of the seizures, especially when of the combined type; marked mental impairment, and the '*facies epileptica*'. Cases presenting the psychical equivalents or the post-paroxysmal psychoses, already described, are also included under this heading; while instances of serial epilepsy and of the status epilepticus are banded together as being most unsatisfactory."

I have made this rather lengthy quotation from Dr. Turner's book because it gives the conclusions reached by one who has devoted many years and much study to the subject of epilepsy. It will be seen that his percentage of curability or great improvement is often higher than twenty per cent. Why is it that the results obtained are so out of proportion to the possibility? One of the chief reasons for this is that the best methods of treatment cannot as a rule be thoroughly carried out under present conditions.

Epilepsy like many other diseases requires a combination of methods in its treatment. These measures in a broad way may be classified as medicinal, dietetic, hygienic (including fresh air, exercise, and recreation), occupational, educational, and surgical. Omitting surgical treatment, which requires particular consideration and which is applicable only in a limited number of cases, of the other measures it may be said, that they are only or chiefly efficient when used in combination. Medicine, without dietetic and hygienic regulation loses one-half of its efficiency, and the same may be said of occupation, and of educational measures. Each therapeutic method needs to be reinforced by the others in order to obtain the best results, and all need to be continued over a long period.

Various reasons might be given in particular cases why systematic treatment of epilepsy is not carried out in ordinary practice, private and hospital. The essential reasons are that the patient often rebels against too long continued treatment of one kind, and that the friends and family not only do this also, but in addition are often not in position to carry out the measures which have been advised. It follows that for the vast majority of epileptics some form of institutional care and treatment must be secured.

Hospital and institutional treatment may be of several kinds.

(1) The state should provide one or more large hospitals and colony farms for those of its wards who are unable, by reason of their financial condition, or that of the persons responsible for them, to pay for care and treatment. The colony farm method has stood the test of time in Germany, Holland, France, Great Britain, and other foreign countries, and also in several of the large states of the Union. The advantages of this method have been ably set forth by numerous recent writers and need not be dwelt upon here.

(2) Semi-private colony farm institutions, like that at Oakbourne, Chester County, Pennsylvania, should be encouraged and aided by the state and by charitable and philanthropic private citizens. Such institutions act as a healthy stimulus to those who work entirely under the auspices of the state. We have many examples of similar institutions for the care of the insane and the feeble-minded.

(3) Small private sanitariums entirely devoted to the care of epileptics, and especially those who offer possibilities of cure, are much needed for epileptics who themselves or through their families can pay freely for what they receive. These sanitariums

should always be well located not too remote from centres of population, and should have not less than ten to twenty acres in connection with the buildings. Preferably the latter should be small, so as to accommodate only a few patients in each cottage; the number of cottages not exceeding five or six. Such cottage sanitarium treatment, thoroughly organized and scientifically pursued, would be a great boon to many epileptics.

(4) In connection with some of our general hospitals and especially with hospitals like those of the University of Pennsylvania, the Jefferson Medical College, and the medical school of the Western Pennsylvania University, should be wards or special buildings given over to epileptics. Such wards or departments should have suburban annexes with a considerable acreage connected with the buildings.

With institutional and hospital methods such as are here outlined, sufficient money for proper maintenance being premised, in the treatment of epilepsy the percentage of cure and of great improvement would be largely increased.

It has already been in part indicated why it is extremely difficult, and often practically impossible to treat epileptics successfully at their own homes. In the case of children many obstacles arise. These depend upon the difficulty of persisting in certain lines of medicinal treatment and in compelling youthful epileptics to follow out more or less rigid dietetic and hygienic measures. It is rare indeed for parents to have the confidence and the patience necessary to carry out a plan of treatment for their epileptic children which reaches over the minimum time limit of two or three years which would be set by every thoughtful physician for a trial of any method of treatment of epilepsy. In my experience in private practice, an experience not without exceptions however, it is difficult to secure absolute acquiescence in a plan of treatment which does not bring about the cure which is earnestly desired in a few months or a year. The patient is taken from one physician to another because in some cases of a recurrence of the attacks, even when these are much diminished, or because of the numerous methods of treatment which are exploited in the press or by friends of the family.

The case is almost equally difficult as regards adult epileptics. Few epileptics who are responsible for their own care will submit to prolonged treatment and to the necessary dietetic and hygienic rules. The cases which come to the outdoor services of our public hospitals frequently go from one dispensary to another, without waiting for results. Not being under special control these patients often violate all the rules laid down for them, including those relating to the medicines ordered. Many of these obstacles would be overcome under the plan of treatment in sanitariums, hospitals, and special institutions, which has been outlined.

It is perhaps impossible to separate entirely the epileptics from the insane in hospitals and institutions for the insane, but so far as possible this should be done. Some forms of epileptic insanity are of such a character that they require care in institutions which are organized under the laws which apply to the insane, but the number of epileptics placed in these hospitals for the insane should be greatly reduced. It is even worthy of consideration whether it would not be better to have a department devoted to insane epileptics in a general institution for epileptics, than wards for the epileptic insane in institutions devoted to the insane.

I am quite positive in my conviction both as regards epileptics and the insane, that the county care of such unfortunates is not desirable, although I know this method has some advocates among the members of this association, and also although much could be, and has been said in its favor with the example of a state like Wisconsin, for instance, before us. Counties like Philadelphia and Allegheny, with a population of a million or more, might provide by special institutions for the care of their own epileptics, as for their own insane. But even in the case of such counties and municipalities, it would in the long run be better for the state to provide special state hospitals for each of its large cities or counties. Conflict between counties, between commissioners and directors of the poor; petty questions of expense; questions as to appointments; the improper use of politics; these indicate some of the directions in which county care of epileptics as well as of the insane, is likely to be not the best.

The little which I shall say in conclusion regarding the treatment of epilepsy by medicinal, dietetic, and hygienic measures, and by occupation and education, will chiefly be to enforce the view that these measures can only be thoroughly carried out in most cases, with the assistance of the machinery of some sort of an institution. Take for example the question of medicinal treatment.

Before the introduction into the practice of medicine by Lowcock about 1857, of bromides in the treatment of epilepsy, the remedies most used were those which may be designated, in accordance with the older nomenclature, as nervines and antispasmodics. Prominent among these were salts of the metals such as the nitrate of silver, the oxide and the valerianate of zinc, and preparations of arsenic. Belladonna was also a favorite remedy much approved and recommended by authorities as great as Trousseau. Undoubtedly some good was achieved by these remedies. Even after the introduction of the bromides, belladonna and the salts of silver and zinc continued to be employed and still have some advocates, especially when used as adjuvants to the bromide treatment and to dietetic and hygienic measures. Whatever medicines are used in the therapeutics of epilepsy, and this applies especially to the use of bromides, it is often good treatment to combine with the most important drug several remedies for particular symptoms and conditions, as digitalis and strophanthus for instance, to maintain the tone of the heart and circulation; choral and antipyrine to add to the sedative efficiency of the bromide, and salol or betanaphthol for the relief of gastro-intestinal fermentation.

The bromides are undoubtedly valuable in the treatment of epilepsy. That they sometimes fail does not argue against their general efficiency. One reason for the complete or partial failure of the bromides alone, or in conjunction with other drugs, is to be looked for in the fact that their use cannot be controlled, modified, and revised except when the patients are under more or less close and continuous supervision, such as cannot be had at their homes or in the wards of a general hospital where such patients usually remain only for a short time. The exact dosage necessary for individual cases can be best determined by institutional observation. In the large colony farms of Germany and other countries, the bromides are still used with much advantage, but their use is not so much a matter of routine as outside of such institutions. Their use is better proportioned to the needs of the individual patient, this being made possible by the fact that he is under continuous observation.

Except in the case of wealthy patients who are willing to establish a thoroughly equipped method of cottage treatment for a single patient, dietetic and hygienic measures are equally, with medical measures, incapable of successful application in the treatment of epilepsy.

Various dietetic measures have been recommended, but with Turner, I believe that the only one of these which seems to promise much benefit is the use of the so-called purin-free* diet, and this or any other plan of diet is of no permanent benefit unless pursued over a long period. Simple as it is I have found it difficult to have it adhered to in private practice. In a well regulated sanitarium or hospital it can be maintained for months or years.

The following citation from Turner indicates what is meant by a purin-free diet.

"The purin bodies, or substances constructed on a base, C N, are widely distributed amongst the common foods.

"Purin bodies exist in all forms of meat extracts, in both the

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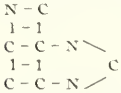
*For those of my audience who may be scientifically interested in the subject of purin-free bodies the following notes with regard to purin, furnished to me by Dr. Edward Lodholz of the physiological department of the University of Pennsylvania, may be of value:

"Nucleo-proteines, a group of compound proteins are found in large amounts in the nucleus of the cell. When broken down they yield simple proteins and a substance known as nuclein. This latter body contains nucleic acid, a body rich in phosphorus and yields purin bases on decomposition. Fisher showed that the purin bases have a common nucleus as follows:



This is called the purin nucleus. A hydrogen compound of this is termed purin. Addition of an atom of oxygen yields hypoxanthin. Addition of two atoms gives xanthin. Addition of three atoms gives uric acid. Numerous other bodies are formed by substitution as guanin, caffein, theobromin. Therefore it is evident that they are intermediate bodies and formed durng the decomposition of nucleus protein."

white and the red meats commonly used as food, and in large quantities in certain glands, notably the thymus and the pancreas. They are also present to a less extent in some vegetables, and in tea, coffee, and cocoa.

"A non-purin dietary may be selected from amongst the following common articles of food: milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, cauliflower, sugar, and fruit, both fresh and dried, and olive oil."

Fresh air, exercises, games, recreation, amusements of a suitable kind would, it is evident, best be secured under some well considered hospital and colony plan.

The subject of occupation in the discussion of epilepsy presents itself in two aspects. In the first place, it is a sad story, that of the epileptic who is compelled by adverse fate, in spite of efforts often courageous and sometimes heroic, the bread earner of a family, to relinquish the occupation for which he has been trained and fitted. For months, or even for years, the occurrence of epileptic attacks is concealed from the employer or others, except members of the family, this sometimes because the seizures are nocturnal. Sooner or later, however, it becomes known that the individual is a sufferer from epileptic attacks, and sooner or later, in the majority of cases, for one reason or another, the occupation is lost, and this often when the patient is still able to do one-half or three-fourths as much work as when he was at his best.

In the second place, the advantages of occupation in the treatment of epilepsy can hardly be over-estimated, if this occupation is of a kind and in the degree which is best adapted to the condition and the capabilities of the epileptic. Occupation which allows of life in the open air during a large part of the time is most desirable, such as is found in gardening and light farming. Here again we see the value of the colony method.

The occupation, whatever it is, whether indoor or outdoor, should be of a suitable and useful kind, and whenever possible money-making. When epileptics in an institution are able to earn money by their continued labor, after reserving an amount sufficient to provide for their maintenance, their earnings should go to their families.

THE GROSS AND HISTOLOGIC FINDINGS IN DEMENTIA PARETICA.*

By N. S. YAWGER, M. D.,
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The structural changes found in dementia paretica are here considered under the headings of the skull, dura, pia-arachnoid, intra-cranial vessels and spaces, cerebrum, cranial nerves, basal ganglia, pons, cerebellum, medulla, spinal cord, cerebrospinal fluid, peripheral nerves, some adventitious substances, and changes in other parts of the body.

Generally speaking, the findings are those observed in advanced cases, though whenever possible the conditions met with earlier in the disease have been noted. Obviously, some observations, as those from studies of the blood and cerebrospinal fluid, must be made for the most part during life. Probably none of the findings in dementia paretica are pathognomonic, therefore, mention is often made of other diseases presenting similar changes. In all, eleven brains were studied, and the tissue was taken as nearly as possible from corresponding areas.

A number of investigators have endeavored to determine an organismal cause for this disease. The most extensive studies in this direction have been made by Ford Robertson in collaboration with others (1) ; these observers stated that a diphtheroid bacillus was the cause of dementia paretica and they described two types of the organism which were called Bacillus paralyticans brevis and Bacillus paralyticans longus. Others (2), in refutation of these observations, have found diphtheroid organisms in different forms of insanity and also in the sane.

Roughly estimated, only about 1 or 2 per cent of syphilitics eventually develop the disease. Since syphilis has almost always preceded dementia paretica, the question is often asked, why is not syphilitic infection more frequently followed by this psychosis? One theory in answer to this is that of Hirschl, who somewhere advanced the view that dementia paretica may develop only in those cases whose near ancestors have been free from syphilitic poisoning and, therefore, may have escaped a partial immunity which otherwise might have been transmitted, had there been a luetic ancestry.

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* From the laboratory of neuro-pathology of the University of Pennsylvania, and the Philadelphia Hospital for the Insane.

The Skull.—The changes here are limited to the calvarium, and the variations observed are in weight, thickness and texture. The weight is practically always increased; this is due to an actual thickening and also to an invasion of the diploë by the compact bony substance. Though this augmentation is most common in dementia paretica, it may likewise be present in epilepsy and paralysis. The increase may be confined to the frontal region or to the occipital region, but more rarely it is diffuse, and occasionally, there is a greater increase on one side. In texture a condensation is shown, which is due to invasion of the diploë by the compact tissue, and this very common condition is spoken of as cranio-sclerosis. To account for the changes in the calvarium, two theories have been advanced; one regarding them as inflammatory in character, and the other considering them due to nutritional disturbances.

The Dura.—On removing the calvarium the dura is often found to be decidedly adherent, but less commonly so than in senile dementia. The tissue is somewhat thickened and vascular, the vessels are tortuous and knotty, the inner surface of the membrane is silvery in appearance and is often marked by fine granulations and rusty stainings; the granulations are most numerous over the sphenoid bone and on the upper surface of the tentorium cerebelli. If death has occurred during a convulsive attack, the membrane will often show considerable congestion. The veins are much enlarged, particularly in the galloping form (3). Owing to an abundant proliferation of the cells in the neighborhood of the vessels (4), an occasional looseness of the dura is observed. Bony growths, either as spiculæ or seed-like in form, are at times met with; their presence seems to have little significance since they are almost as common in the sane; they are usually found in the falx. Adhesions are occasionally observed, however; these are present normally in the area adjacent to the superior longitudinal sinus. The subdural space is occupied by an increased amount of clear or cloudy cerebrospinal fluid. A subdural false membrane is sometimes present and this is more frequent in dementia paretica than in any other psychosis; the most common sites of this membrane are the sides of the vault and in the middle fossa at the base; it may occur on one side or on both; about its production considerable controversial literature exists. This formation has been studied most extensively perhaps by Ford Robertson (5), who recognizes two types depending upon their origin—one, as the direct outcome of a morbid process in the dura itself, and the other, as the result of hemorrhage into the subdural space, which has taken place independently of any morbid condition within the dura. Within the subdural space, the so-called meningeal hemorrhage may have occurred, and it may be in the form of a thin film of blood, or a thick coagulum; sometimes it has undergone organization and perhaps may show cyst-like formations. It is worthy of note that these hemorrhages now are said to be less common since falls do not occur so frequently, because of the more careful supervision of the insane. Microscopically, the more important changes to be noted are an increase in fibrous tissue together with the changes in the endothelial cells, and these are both productive and degenerative in character. Granulations are formed and there are at times aggregations in the perivascular channels which may be mistaken for round-cell infiltration. Other cellular changes are vacuolization and fatty and hyaline degeneration. Minute hemorrhages occur within the membrane and many new capillaries are formed. Hyaline areas may be seen as may other substances whose positive chemical nature has not yet been determined.

The Pia-arachnoid.—Conditions affecting this membrane are at once seen to be serious when it is recalled that through the meshes of the pia flows all of the return blood and lymph of the cerebral tissue, and a marked retardation of this flow results in atrophy of the brain substance. On exposure this membrane may float up from an excessive accumulation of fluid underneath. Cysts are frequently seen and small hemorrhages often may be observed. A more or less widespread cloudiness of the tissue is present; to a lesser extent this is common in advanced life, and it is always present in chronic alcoholic insanity and in senile dementia; however, this cloudiness is more extensive in dementia paretica than in any other psychosis. In cases of more than a year or so standing there are usually present variously sized dilated and congested vessels which lie in a thickened, tough, milky-colored, or opaque membrane, and these appearances are most marked over the sulci and between the convolutions, they are likewise much more apparent over the convex surface of the brain except in the occipital region, also over the inner surface, but less noticeably so over the basalar area; in places, the convolutions may be almost indiscernable. Occasionally, the changes in the soft membranes are particularly pronounced in the occipital region, but this is usually observed in those cases which begin as tabes. The Pacchionian bodies not infrequently show noticeable enlargement and in their neighborhood the membrane is particularly apt to be thickened. On removal of the membrane just after death, a blood-stained turbulent fluid oozes out. The superficial and deep surface is everywhere injected and even the cortex shows this condition. At times the pia-arachnoid is noticeably adherent to the cortex, and when stripped off, small parts of the brain substance are found attached to it. According to Mott (6), if the membrane is removed before rigor mortis sets in, it may be taken off with ease and without leaving erosions on the cortex. If a normal brain is allowed to undergo post-mortem change, there will be softening of the cortex and particles will adhere to the membrane when it is removed, therefore, these adhesions do not have the pathologic significance that has at times been bestowed upon them.

Microscopically, we find the arachnoid space lessened and in some areas obliterated. There is an increase of the tissue of the trabecular framework, a cell infiltration, to which is probably added proliferation of the endothelial cells; these little round cells have a relatively large nucleus surrounded by a small area of protoplasm; in many instances they are exceedingly numerous, packed within the irregular channels of the membrane, and they are also conspicuous about the arteries and veins, ofttimes forming a sheath about them. In cases of long standing this appearance is partly lost through disintegration and absorption. Three varieties of cells are conspicuous in this tissue; these are plasma cells, mast cells, and lymphocytes. Trypanosomiasis also gives a widespread lymphocyte and plasma cell meningeal infiltration.

Cell disintegration is often observed; it may be pigmentary or hyaline in character; small osteoid plates are at times met with; granules of hematoidin may be seen, and granular débris, partly the result of disintegration of red blood cells and also due to breaking down of leucocytes and endothelial cells, may be observed.

The Intracranial Vessels and Spaces.—That the small vessels of the pia-arachnoid consist only of arterioles and venules, without capillaries, is the contention of Ford Robertson (7). As the disease develops, the blood-vessels become more numerous. One of the earliest changes observed is the appearance of round cells in the connective tissue sheaths of the arteries and veins; later, the entire adventitia may be invaded and even distended by these elements, the vessels are often completely ensheathed. One of the most striking features of the microscopic appearance of the cortex is the great number of small cells about the arteries, capillaries and veins; among these are to be found plasma cells which are always present in dementia paretica; the belief appears to be growing that many of these perivascular cells are plasma cells; however, just what a plasma cell includes, is a mooted question. There seem to be many atypical forms, others are not mature, and often they are undergoing deterioration; it may be for these reasons that so much difference of opinion exists. They seem to have been first described by Unna, who demonstrated their presence in the skin, and who considered them to have a local origin; he believed they were hypertrophic connective tissue cells. Marscholko regarded them as emigrated and transformed lymphocytes. They may often be seen to ensheath the vessels, around which it has been said "they cluster densely, like plant-lice upon a young shoot." Often they are found in the dilated adventitial lymphspaces of the vessels of the cortical and subcortical tissue; they occur over the whole cortex, in the central ganglia, and in the cerebellum. Plasma cells have also been described as occurring within the cerebral tissue; the possibility of this being an error is pointed out by Evenson (8), when he says, "they may belong to a capillary, whose wall has not been included in the section, or which has not been stained for some cause or other." As commonly seen, the cell is large, is more or less rounded, angular, or slightly irregular in outline, contains finely granuled protoplasm surrounding a usually more or less eccentrically placed wheel-like nucleus, just beyond which may be seen a clear area or sort of halo; sometimes a faintly stained nucleolus may be observed. The cells stain well with both theonin and toluidin blue, and, according to Unna, are often present where there is irritation of the connective tissue cells, especially in the immediate vicinity of the blood-vessels in chronic inflammations. Their function seems to be to aid in vascularization and cicatrization. Plasma cells frequently show degeneration and vacuolization, they are usually confined to the adventitial lymphspaces, and only extend beyond this in parts near an adherent pia, or where there is intense infiltration. The same remark applies to the distribution of lymphocytes, with this difference, that these cells occur more frequently in the cells of the large vessels, and not, as do the plasma cells, in the walls of the small vessels. Most cells are spherical or ovoid in shape, with coarse basophile granules, and a large, oval, eccentrically-placed nucleus, which is badly marked off from the surrounding protoplasm; they always occur discretely. The most distinctive microscopic sign of dementia paretica is the widespread adventitial plasma cell infiltration. Plasma cells are also numerous in the general body organs. Trypanosomiasis is another chronic disease which gives considerable leucocyte and plasma cell infiltration in the perivascular spaces. Yellow pigmentary deposits may be seen in the adventitial coats of the cerebral vessels, but not quite so frequently as are seen in senile insanity. They have been taken for hematogenous matter, but may be distinguished from this substance by their paler color and wax-like appearance; occasionally, however, it may be impossible to differentiate them, as chemical tests are often unsatisfactory. The media is frequently seen to have undergone hyaline change, the muscle nuclei may scarcely show, and at times there is considerable cell infiltration. Under examination with osmic acid, the muscular tissue may show fatty degeneration; there may be a little thickening of the intima. In cases of long standing, these changes have undergone some disintegration, and partial absorption has ensued. The arteries of the pia-archnoid, as well as those of the intra-cortical tissue, are involved, and the veins are also implicated. A similar picture is present in cerebral syphilis, and in both psychoses many of the arterioles of the pia and cerebrum show periartritis; that is, a localized and condensed infiltration of the adventitial coats with leucocytes; proliferation of the connective tissue cells should not be mistaken for this condition. There is widespread hyaline, fibroid degeneration of the vessels of the brain, and pia-arachnoid. The capillaries of the cortex and white matter are thickened and cellular in areas. Local obliterations may cause softening and atrophy. So-called colloid degeneration of the brain extensively implicating the vessels has been carefully studied in one case reported by Alzheimer (9). According to Evenson (10), the large cerebral arteries, especially at the base, often appear rigid, gaping widely when cut across, and are dotted over with white or yellow spots; similar dottings are seen in senility. Between the endothelial layer and the elastic membrane of the intima of the larger arteries there may be observed another irregular layer made up of a fibrillary ground-work, within the meshes of which there are many cells. The elastic membrane is irregular or ragged. The muscular coat is but little affected, though fatty degeneration is at times demonstrable with osmic acid. The adventitia is thickened and often contains leucocytes and plasma cells.

The Intracranial Lymphspaces and Channels.—Strictly speaking, there are no distinct lymphatic vessels as in other parts of the body; there are large and small spaces, and channels; the most diminutive of the latter are the intercapillary channels. The lateral ventricles are usually dilated and at times are markedly distended. Granulations of the ependymal lining are almost always present; these are considered under the neurogliar changes. After passing through the capillary walls, the lymph penetrates the small spaces of the intervascular tissue and then passes into the adventitial channels of the arterioles and venules. In many of the spaces of the pia-arachnoid and cortex, there is a round-cell invasion.

The Cerebrum.—There is considerable wasting of the brain and this is at times observable before the dura is opened; it is said (11) that on an average the brain is 152 grams below its normal weight. This loss is much greater in cases of slow evolution. Heavy brains have been reported, but it is doubtful if these were really cases of paretica dementia. The loss in weight is not equal on the two sides, the right hemisphere usually being the heavier, and the difference may be as much as 50 grams. The general atrophy is most evident in the frontal and central regions. There is flattening of the convolutions, widening of the sulci, a thinning of the cortex, which latter is opaque and injected. Section of the brain shows more or less œdema and the tissue is softer than normal; there is usually congestion and the layers are indistinct. Recent hemorrhage is often observed and atrophic areas of softening in the cortex are sometimes met with. At times there may be seen small stripes or spots which mark the line between the cortex and white matter; these are due to destruction of the fibers of Tuckek. According to Nissl, there are no gummata found in dementia paretica and in this connection Mott says it is very rare to find extensive endarteritis or gummatous syphilitic lesions in true general paralysis.

Atypical cases are sometimes met with and the disease may be ushered in by a hemianopsia; this may occur when the psychosis begins with extensive involvement of the occipital lobes. Sometimes there is an early apoplexy and focal symptoms have been known to precede the usual manifestations of dementia paretica, an occurrence which at times leads to a wrong diagnosis.

The nature of the histologic changes has been explained differently at different times. At first it was thought to be inflammatory in character; then a systemic degeneration of certain fiber tracts was considered the essential cause of the pathologic changes; at present the most common belief is that of a primary nerve element degeneration due to some toxic substance. Changes are seen in the nerve cells, nerve fibers, and in the neuroglia. The marked proliferation of blood-vessels and other vascular changes have already been considered. There is disorder and a more or less noticeable disappearance of some of the nerve cells. The cortical layers are difficult to distinguish and at times cannot be differentiated; this disarrangement of the cortical strata Nissl regards as the most noticeable and only distinctive anatomical feature of dementia paretica. The cell body is reduced in size; it is granular and pigmented; some of the processes have disappeared and the angles are less noticeable. Changes or destruction have taken place in the Nissl bodies; the cells may be hyaline in appearance or there may be granular substance more or less diffusely distributed throughout the body of the cell. The nucleus is darker than normal, it is often diminished in size and sometimes appears angular; the nucleolus has also become smaller and has approached the nuclear boundary. Yellow pigmentation may be seen in many of the small cells; this never occurs normally, even in the aged.

The neuro-fibrillary changes have been studied by Schaffer (12) with the employment of Bielschowsky's silver impregnation and method; he found that all areas examined showed changes; those observed early were most noticeable in the central gyri, comparison of the cells in the paracentral lobule with those in the normal brain, showed swelling of the cell body, a diffuse staining of the apical processes and the reticulum was well preserved; often the fibrils were disintegrated and the intervening substance deeply stained. In other areas, the reticulum showed marked disintegration; sometimes there was just a disappearance of the fibrils around the nucleus. Some cells may show evidence of acute disease manifested by their being swollen and taking the stain less freely than normally; the nucleus is swollen and approximates the edge of the cell; vacuoles may appear.

As regards nerve fibers, many are destroyed; the projection fibers, as well as those concerned in association, show degeneration. The tangental fibers of the surface are usually the first to show change. The medullary tubing is at times entirely lost. Many parts of the brain are affected, but the changes are most extensive where atrophy, cellular, and vascular changes are most marked.

Trypanosome disease also shows slight nerve-cell and nerve-fiber destruction.

In the neuroglia there is an extensive overgrowth, especially in the superficial layers of the cortex where the fibers may penetrate the pia. The glial overgrowth is also especially marked about the cortical vessels and even in the white matter. Quite characteristic is the presence of giant cells, and a rich formation of thick fibers which are attached in bundles to the blood-vessels. The Stäbchenzelle of Nissl (13) is a long cell with a slender and an occasionally bent nucleus, the latter often being broken or shrunken; this cell may be branched and at times contains fat and pigment. Nissl believes these staff-like cells to be of neurogliar origin; they are often found near the adventitia of the capillaries and may have something to do with the production of these vessels. In dementia paretica they are present to a degree not found in any other disease. Granulation of the ependyma is a lesion that has received but little attention in this country; these granules are at times so thickly studded as to present the appearance of fine sand paper; they are often as large as grains of granulated sugar, and are most common in the lower half of the fourth ventricle, where they are seldom wanting. Baird (14), in 262 examinations, found them present in more than 90 per cent of the cases; they occur very early in the disease and he considers them the most characteristic naked eye sign in dementia paretica; they are not uncommon in senility and are sometimes met with in hydrocephalus; many cases of senile dementia show the condition. One explanation of their origin is that of epithelial proliferation, but their most probable derivation is that of a neurogliar overgrowth, and it seems likely that they result from irritation produced by products in the cerebrospinal fluid; these latter substances Mott (15) suggests may be due to a complex lipoid as well as a specific globulin increase, as the process of neuronic decay proceeds.

The Cranial Nerves.—In the nuclei of the cranial nerves the ganglion cells are affected by changes similar to those found in the cells of the cerebral cortex.

The optic nerve is the one most frequently affected and the changes here usually occur in the group of cases known as taboparalytics. The primary seat of the disease is probably in the retina. The atrophy is more marked towards the globe and deminishes towards the cerebrum. The retina shows degenerative changes in the ganglion cells and the nerve fibers; the vessels of the nerve show thickening and degeneration. In the nerve fibers of the optic nerve, there is breaking up of the myelin and a varicose condition of the axis-cylinders.

The vagi are frequently diseased and this has been regarded as conducive to tuberculosis of the lungs (16), a disease which quite commonly develops in dementia paretica. The ascending root of the fifth nerve is often sclerosed in the medulla. Other nerves which sometimes show changes are the olfactory, oculomotor, and hypoglossal. The cranial nuclei of the trifacial, facial, and hypoglossal nerves are frequently involved.

The Basal Ganglia.—Here are found changes similar, but less extensive than those observed in the cortex; there is destruction of some of the cells, a more or less loss of nerve fibers and proliferation of the neuroglia.

The Pons.—There is congestion, and areas of softening and atrophy are at times met with. The soft membranes may be adherent and they frequently contain dilated vessels.

The Cerebellum.—The changes here have been carefully studied by Sträussler (17), who states that the microscopic appearances of the disease are less pronounced than in the cerebrum. There is less meningeal thickening and the vascular changes are not so marked. Frequently, vacuolization and some loss of nerve cells are met with. The fibers are decreased; there is neurogliar proliferation and a shrinkage of the convolutions. The tissue most affected is about the semilunar sulcus, central lobe, superior lobe, inferior lobe, the nodes, and the vulva. Sometimes the cells of Purkinge are surrounded by a dense basketwork of neurogliar fibers, and the fibers of Bergman may be evident in the molecular stratum. Normally, there are no neurogliar fibers present in the granular layer, but now they may be demonstrated.

The Medulla.—Slight opacity of the surrounding membrane is sometimes met with. The nuclei of origin of the pneumogastric nerves at times show changes, and, more rarely, other nuclei do also. There is some nerve-cell disturbance and a neurogliar cell increase is observed.

The Spinal Cord.—It is pretty generally accepted that the spinal cord shows more or less change in dementia paretica. In nearly half of the cases the membranes are thickened and adherent, and there is visible shrinkage of the posterior and lateral columns. The lesions may be either diffuse or systemic. The nerve cells have undergone degeneration and atrophic changes similar to those observed in the cerebral cells. Neuro-fibrillary abnormalities have been observed here similar to those seen by Schaffer in the cells of the cerebral cortex. The microscopic changes are most frequent in the posterior ascending tracts; they are similar to, though not so extensive, as those seen in tabes. The nerve fibers show two types of lesions; (a) the tabetic type, where the degeneration is localized to the posterior columns; according to Ribaud, who examined many sections, the lesions of the posterior columns are not strictly systemic, but are characterized by irregularity, which is evident from examinations of sections taken at different levels of the cord, by their diffuseness apparent in a single section of the cord, and by the frequent coexistence of spinal sclerosis with an intact condition of the roots of the zone of Lissauer; (b) combined sclerosis, where the degeneration involves both the posterior and the lateral columns.

It has been stated by Nagette, Möbius, Mott, and others, that dementia paretica and tabes are the same disease, a metasyphilitic affection in which the brunt of the disease falls in one instance upon the brain, and in the other upon the cord. In some cases both diseases seem to progress together; generally speaking, however, the psychosis has preceded the disease of the cord; these cases constitute the tabo-paralysis group.

The Cerebrospinal Fluid.—At the present time so much work is done upon this subject that additional new and important matter may be brought out at any moment. As yet, the psychiatrist is only interested in the pressure, the proteid content, and the cellular elements; all other data is only of academic interest. The acidity or alkalinity, and the presence of a copper-reducing substance, seem so far of no practical value, although the latter substance is said by Pike (18) to be absent in dementia paretica; upon what authority this statement is made, I do not know. The fluid is always greatly increased in amount. The hydrostatic pressure, which Krönig (19) found by doing a lumbar puncture on a healthy adult person in the horizontal position to be 125 mm., is very much raised, possibly several times above the normal. The proteid content is always increased; it is a globulin which normally does not exceed a half-gram per liter, but in dementia paretica it may be several times that amount. The globulin is of a special variety known as euglobulin and is said to carry the peculiar Wasserman anti-body. In the serum of cases of dementia paretica are usually found substances resulting from the destruction of tissue; these are lipoids and globulin and are termed anti-bodies; it is these substances upon which so much laboratory work is at present being done; this matter will only be touched upon here. The reactions and tests most studied are those of Wasserman, Noguchi, and Ross-Jones.

The Wasserman reaction, which is applicable to both blood serum and cerebrospinal fluid, was suggested basically by the property of hemolysis observed when the blood serum of one animal dissolves the red blood corpuscles of the blood of an animal of another species with which it is brought in contact. The reaction depends upon the presence of three substances known as compliment, which is always present in the blood serum; anti-body, which results from the reaction of the injected animal against the injected red blood cells; and antigen, which is the injected blood corpuscles. Syphilis and certain other diseases produce anti-bodies.

The technique of this reaction is sufficiently difficult to require for its application one skilled in this special kind of laboratory work. Noguchi has modified the test so that dried reagents may be used. It has been stated (20) that a positive Wasserman reaction in the blood with a positive reaction in the cerebrospinal fluid, and a lymphocytosis, points to a parasyphilitic condition of the central nervous system.

Unfortunately, it is in the early cases that the Wasserman reaction is most apt to fail, and sometimes it also fails in advanced cases, but from recent observations it seems that over 90 per cent of these patients give a positive result. The amount of so-called compliment deviation may be used as an index to the activity and duration of the disease.

The Noguchi butyric acid test depends upon the production of a flocculent precipitate when equal parts of a blood-free cerebrospinal fluid and 10 per cent butyric acid in normal saline solution are boiled, to which normal caustic soda solution is afterwards added, and the whole is then boiled briefly. The action is upon the globulin. A positive result is sometimes gotten in tertiary syphilis, and all forms of meningitis give the reaction.

The Ross-Jones reaction depends upon the presence of an *excess* of globulin, which forms a thin white film at the junction of cerebrospinal fluid with a saturated solution of ammonium sulphate. Turner (21) states that this reaction was only positive in general paralysis and cerebral syphilis. An additional advantage is that it gives the reaction early in the disease. In 48 cases of general paralysis, or suspected general paralysis, he obtained only two negative results; one was a case of seven years' standing, and the other was a recent and doubtful case. The latest investigations seem to show that this reaction is only positive in dementia paretica, tabes, tertiary syphilis, and in syphilis of the nervous system.

The Cellular Elements.—Normally, only three or four cells are found to the cubic millimeter and these are exclusively endothelial cells, although the fluid after being drawn and centrifugalized may show the presence of a few erythrocytes which, however, are inconsequential. The counting is facilitated by the addition of a trace of methol violet solution, which imparts a pale tinge to the leucocytes. By cytosis is understood the presence of five or more cells per cubic millimeter; more than a hundred are at times present. The increase begins at the very onset of the disease and is more marked in the acute cases. The cytosis is especially greater as regards the polymorphonuclear leucocytes, during the seizures that occur in the disease. Most of the cells are lymphocytes, usually two-thirds are also endothelial cells, phagocytes and polymorphonuclear leucocytes. It is desirable to differentiate between endothelial cells and plasma cells. Lymphocytosis does not diminish with antisyphilitic treatment and this will distinguish it from pseudo-paralysis.

The Peripheral Nerves.—Lesions here consist in the phenomena of neuritis and atrophy analogous to those encountered in tabes and alcoholism; that is, the parenchymatous degeneration with some overgrowth of connective tissue; both motor and sensory nerves are involved.

Some Adventitious Substances.—Bodies of various sizes and shapes, whose positive chemical nature it is difficult to determine, are occasionally met with. A case of colloid degeneration of the brain extensively implicating the vessels, has been described by Alzheimer (9). Colloid bodies are at times observed in the outermost layers of the cortex and are ascribed to degeneration of the nerve fibers. They have been seen in the medulla in locomotor ataxia. (22). The writer (23) has himself studied a case of dementia paretica that had sustained repeated and severe blows to the head 20 hours before death, in which colloid bodies were found in the brain in great profusion; these bodies were not regarded as the result of the psychosis, but seemed to have resulted from rupture of the myelin sheaths with protrusion of particles of myelin; these particles responded to the tests usually applied for determining the presence of colloid bodies.

Osteod plates are sometimes observed; they are small and may occur either as spiculæ or they may be seed-like in form; they are met with most frequently in the falx, but occasionally are seen elsewhere in the dura; they probably have little significance since they are almost as common in the sane.

Changes in Other Parts of the Body.—Unless the disease is cut short through some intercurrent affection, the patient usually dies of asthenia. In the second stage of the disease, there is usually considerable fat formation, but ultimately the emaciation is extreme; bed-sores, which are usually present, may be of enormous extent, and they not infrequently give rise to a fatal septicæmia. There may be somatic evidence of syphilis in the form of scars on the penis, thighs, and legs, and there may be nodes along the tibiæ. The cutaneous evidence of former syphilitic lesions is often wanting, and in this connection it is interesting to recall that by some, notably Fournier, it has been contended that in many cases of dementia paretica, the manifestations of the preceding syphilis have been mild in character. A neuropathic arthritis is occasionally observed in those cases attended by tabes. It differs from the common form of arthritis in being more acute, and is confined for the most part to the large joints, usually the knee. Without previous injury, the joint suddenly swells and an infusion forms which lasts for a variable time; ulceration sometimes results, new formations may develop, and displacements take place. Pain is noticeably absent. This peculiar form of arthritis has been thought to be due to changes in the anterior horns of the cord. Hematoma auris is at times present, but there is probably nothing in this condition to warrant any connection with insane states other than its development being favored in persons of lowered vitality; it occurs for the most part because of the vulnerable position of the ears, and also because of the violent tossing of the head where motor restlessness is a marked feature of a psychosis. Instances of its occurrences in normal persons are seen in injuries sustained while playing foot-ball and in other rough sports. Absorption is more common in normal persons, as in these the nutritive processes are acting more favorably, and besides better care is taken of the injured part. In the osseous system, as has been previously observed, the calvarium is thicker and denser than in the normal state, and these changes are distinctly different from those found in bones in other parts of the body, changes are particularly noticeable in the clavicle, femur, and ribs, and in the latter the changes at times are so marked as to permit of easy fracture between the thumb and fingers. The bones have become brittle and there has been softening and absorption in their interior; a diminution of the bone marrow is also noticeable. On microscopic examination, the bones have been described (24) as showing a granular condition of the laminæ, particularly around the Haversian canals, which latter are large, unequal, irregularly dilated, filled with opaque matter and a few oil globules. It is at times unjust to assume that fracture is the result of rough handling on the part of attendants; in addition to brittleness of the bones, these patients do not guard against danger, they are ataxic and more or less insensible to pain, and muscular action alone is at times sufficient to cause fracture.

In the vascular system marked changes are observed. There is often atrophy of the heart and valvular lesions are sometimes met with. A most common condition is that of atheroma of the aorta; in 69 cases out of 84, Straub observed that the aorta showed signs of a syphilitic process, which he maintained was different from the atheroma of old persons. Brodington (25), from studies of the blood, found the polymorphous cells reduced in the second stage, while in the mononuclears were increased; in the third stage this was reversed. In both stages the lymphocytes were less abundant than the hyaline cells. Eosinophils were usually more numerous in the second stage, as were the mast cells, when they were found at all. Myelocytes were only occasionally seen. During convulsive seizures there was marked leucocytosis and sometimes the increase was from four to six-fold in a few hours; the increase was in the polymorphous form; hyaline cells and myelocytes were increased. The blood in the third stage was dark, viscid, and flowed with difficulty, the reds were high and the hemoglobin and specific gravity were reduced. In the third stage the blood pressure was high during excitement and low in the condition of depression.

Idelsohn (26) has observed a reduction and perhaps loss in the bacteriacidal properties of the blood. The autovirulence is increased according to D'Abundo (27). An increase of lecithin is also observed in the blood.

The general organs of seven cases were studied by Catola (28), who found arteriosclerotic changes, capillary hyperæmia, interstitial hemorrhages, cloudy swelling, connective tissue hyperplasia, and fatty degeneration. There was a diffuse distribution of plasma cells in all organs studied, and this, the writer believes, points to dementia paretica in the absence of other special lesions to account for them.

At autopsy the lungs frequently show pneumonia and gangrene, and also tuberculosis, and this latter is said to be particularly common when the vagi are diseased.

In the urinary system there is atrophy of the kidneys in about one-third of the cases. Barnes (29), who studied the urine in five cases, summarized his observations as follows: (1) A tendency towards a low total nitrogen; (2) the undetermined rest is high and this in part is explained by the fact that all urines except one showed traces of albumin with trichloracetic acid, and that in these a few granular casts were to be made out with the microscope; (3) the neutral sulphur is in the majority of instances high, while the etherial is low. There appeared to be wide daily variations in the amounts of the urinary constituents secreted. Ball has stated that about 7 per cent of the cases show a trace of sugar. Blumenthal (30) has found an increase of lecithin in the feces. The changes in the muscles were studied some years ago by Campbell (31), who found an extreme degree of atrophy, partial or complete disappearance of a number of muscle fibers, an increase of the nuclei of the sarcolema, and a cellular infiltration of the interstitial substance; there was a lessening in the number of the motor end-plates and some showed degeneration. He also claimed that he could demonstrate the existence of most pronounced changes in the muscular elements of the heart in cases where the vagi were diseased. This is at variance with the more recent studies of Barratt (32), who examined the vagi and sympathetic nerves from ten cases; he found no evidence of degeneration of the medullated nerves, nor was there any alteration in the connective tissue surrounding the nerve fibers; the sympathetic nerves examined were the cardiac branches of the vagi and the nerves around the coronary and splenic arteries; these showed no abnormality. This observer confirmed the previously expressed opinion of Mott "that the fatty degeneration of the heart and other muscular structures, which he found in general paralysis, could not be accounted for by recognizable degenerative changes in the vagus or sympathetic nerves.

In studies of body temperature, Coleburn (33) found that early cases showed no appreciable departure from the normal; late in the disease emotional excitement, epileptiform, and paralytic seizures, and minor physical disturbances produced effects upon temperature out of proportion to the causes, and elevations unaccompanied by leucocytosis occurred without assignable physical basis.

Juvenile and adolescent paresis are terms usually employed to designate cases which develop dementia paretica after having had congenital syphilis, or in persons where there has been luetic infection early in life. Although numerous cases have now been reported, it must be that the disease is often overlooked in this country, since many large institutions show no record of such cases. The disease has been observed at least as early as eight years of age, and some of the cases studied have been as old as 33. The pathologic changes seem to be identical with those found in the adult.

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Trauma as a Cause of Amyotrophic Lateral Sclerosis

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TRAUMA AS A CAUSE OF AMYOTROPHIC LATERAL SCLEROSIS

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The following cases have been studied with a view to estimating the correct relation of the trauma which in each case preceded the symptoms of amyotrophic lateral sclerosis.

Case 1.—I examined a mechanic, aged 44, in the dispensary of the University Hospital in 1909. Five years before that date he had fallen 12 feet and injured his back sufficiently to keep him in a hospital for two months. After four years of normal health he caught his right hand in the machine which he tended, badly tearing the soft tissues, and again was forced to spend two months in a hospital. On resuming work, he immediately felt that the injured hand was weak, awkward and easily fatigued. The lameness and ensuing atrophy spread up the limb to the shoulder, so that within twelve months the right upper extremity was nearly useless, while the left hand had begun to show the early symptoms of the disease. The picture of amyotrophic lateral sclerosis rapidly developed and the patient died early this year with bulbar paralysis.

Case 2.—A railroad engineer recently in Professor Soiller's service at the University Hospital attempted just a year ago, after a day's work, to jump on a moving train. He lost his grip and for a moment was in danger of being killed; but by a great effort he regained his hold and managed to get on the car. He lay for the rest of the two-mile run much agitated and violently trembling in his legs. Next day, however, he was able to walk and returned to work, and for two weeks all went well. Then he felt stiff and sore in the muscles of the lower limbs, and this condition progressed as time passed. Five months later, again while returning home from work, he tripped on a cross-tie and fell on a bridge, striking his left shoulder as he passed through, finally lighting on a girder five feet below. He was able, nevertheless, to draw himself back to the track and to walk home. The next day he felt dizzy and nauseated. After a few days, weakness and lameness developed in the left arm, followed by atrophy, the symptoms increasing from day to day until by October, 1910, the patient was forced to give up work. He has now the cardinal symptoms of amyotrophic lateral sclerosis.

Among the records at the Philadelphia General Hospital I find but few case histories in which the diagnosis of this disease has been made. Among the symptoms in several cases of spinal muscular atrophy are recorded spasticity of the legs, and some cases of lateral sclerosis showed muscular wasting in the arms. In two out of the whole list accident had preceded the disease:

Case 3.—Two years before onset the patient had fallen from a moderate height. His first discomfort was a stinging paresthesia in the toes, felt several months after the injury. Later general muscular wasting appeared, together with spasticity of both legs.

Case 4.—The patient five months before the onset had fallen from a height of 25 feet and been rendered unconscious.

Case 5.—The case reported by Dr. George Price[1] happens to be exactly apposite. A middle-aged acrobat made a false step and sprained one ankle. Shortly afterward he sustained a slight injury from a fall. Within a short time muscular wasting appeared with some stiffness and slightly exaggerated tendon reflexes in both legs.

Gelma and Stroehlin in a report[2] on this subject tell of a workman who received a severe strain of the vertebral column while trying to support a 700 kg. stone which was slipping from the hold of a derrick. He felt weakness at once. Stiffness and atrophy succeeded and the disease went on to bulbar paralysis within three years.

Kirk Mendel[3] discusses the etiologic relation of trauma to amyotrophic lateral sclerosis, and reviews the cases reported up to that time. A patient of his, a woman, had fallen from a height of ten feet, striking her left side. Seven months later she again fell, this time from a hay-wagon, bruising her left breast. Two months later marked fatigue was felt, followed by numbness of the left upper extremity and atrophy. The symptoms were earliest and throughout most marked in the left upper extremity.

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[1] At the session of the Philadelphia Neurological Society at which this paper was read.

[2] Gelma and Stroehlin: Gaz. d. hôp., March 23, 1911.

[3] Mendel, Kirk: Umfall in der Aetiologie der Nervenkrankheiten, Berlin, 1911.

Valentiner's patient showed symptoms shortly after a fall during a violent athletic effort. Goldberg tells of a workman who fell on his buttocks from a scaffolding, and whose right foot was crushed by an after-falling tool-box. The symptoms started in the right foot and were always more severe in the right leg. Hauck's patient received a deep cut in the left forearm and right leg, and here also the injured limbs showed earliest and most severe signs of the disease. The man reported by Ottendorf had hung suspended by arms and chin for several minutes from a beam. Seven months later the disease commenced in the arms and progressed to bulbar palsy. Pagenstecher almost duplicates this history, except that his patient showed immediate symptoms. An electric shock preceded the onset in a 6-year-old boy examined by Hoehl. Oppenheim, in his text-book on nervous diseases, tells of a man who after an accident on the water was compelled to row violently to save his life.

Gowers[4] deals with spinal muscular atrophy and amyotrophic lateral sclerosis without distinction. In a few cases, he says, he has known the complex to develop after a concussion "as if it had set up a perverse nutrition of the nerve elements. Still more rarely a fall injuring one limb has been followed by muscular atrophy commencing in this limb."

Amyotrophic lateral sclerosis has been reported as following parturition.

It thus appears that many patients in whom this relatively rare spinal degeneration has occurred refer it back to some kind of trauma. If these accidents had been of the slight kind which most workmen suffer every year or so, the question of causal relation might be dismissed. But the injuries were in each case unusual, so that it is difficult to dismiss them as mere incidental antecedents. Mendel calls attention, as did Gowers, to the fact that the disease often develops first and persists most markedly in the injured member, or if not in the same limb, then on the same side of the body. In Mendel's opinion the interval elapsing between injury and early symptoms is sufficiently constant to merit consideration.

Many writers on this disease do not even mention trauma as an etiologic factor. Winterscheid thinks it possibly a proximal cause, and this is Mendel's conclusion, who feels that some congenital defect must be invoked to explain the tendency to the degeneration. The question comes up, What is there about the circumstances attending such accidents that could thus operate on the spinal cord? Two possible mechanisms suggest themselves. If the blood-supply to certain tracts and cells were congenitally precarious, a severe concussion might completely cut off part or all the nutrition of the parts involved. It must also be remembered that violent exertion usually accompanies the accidents above described, and, as Edinger has suggested in regard to tabes dorsalis, overfunctioning might undo an already weakened tract. In the various forms of professional neuritis overstimulation of nervous cells is the most obvious cause of the changes in structure.

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[4] Gowers: Text-Book on Nervous System, i, 473.

Before attempting to reach a conclusion from these cases as to the causative value of trauma, there is another possible relationship between the disease and the accident that is worthy of consideration. The accident might be the result of the disease. A mechanic whose hand was gradually losing its wonted deftness through an incipient central degeneration in the cervical cord one day fails to remove his hand in time from his machine; an engineer, unconscious as yet of weakness in hand and leg, tries the muscles too far in the effort to jump on a moving train. The workman whose stiffness has not shown itself in ordinary walking has the defect painfully emphasized when a slight misstep throws him from a beam in midair. Siccard in discussing one of the cases above reported took a non-committal position because he could not decide from the evidence that the preexisting disease had not caused the trauma.

Patients suffering from any insidious disease are apt to overlook the early dawning of their trouble. Close questioning often enables the physician to place the onset weeks or months earlier than the patient had dated it. Very often, too, the patient assigns some unusual event as the starting-point, or even as the cause, of his illness—explaining malaria as a result of a wedding; or apoplexy, of a comet. If patients with half of each retina totally blind can walk the streets and do daily work for months oblivious of the defect until a collision with a street-car calls it to their attention, it is not hard to believe that slight, incipient lateral tract disease may escape notice until it produces sufficient awkwardness of movement to cause an accident.

The gradually developing maladroitness would be accentuated by weariness and thus increase the liability to mishap at such times. Furthermore, rest, such as is enforced for a period after the accident, would render the beginning motor defect less noticeable, while resumption of work after the immediate effects of the injury had disappeared would fix attention on the injured member and call into prominence its defect in power and dexterity. This accounts for a somewhat definite period that elapses between the trauma and the early symptoms supposed to result from it.

But after giving due weight to this reversal of causative relationship, and having subtracted all cases in which uncertainty remains, a certain number of cases lie before us in which no agency in producing the accident can be charged to the patient himself. The trauma acting on parturient women, the electric shock received by the boy, and the injuries received by the member first striking the ground, in patients who fell from buildings, cannot be charged to preexistent disease in the sufferers. Here we must conclude either that the trauma called forth the disease, or that the trauma preceded it merely as a chance antecedent. The attendant circumstances in some of the cases are such as to make the theory of chance improbable, though a much larger number of cases must be presented before it can be definitely eliminated.

The histories here presented admit, therefore, of one conclusion: A reasonable presumption exists, but no definite proof, that trauma is a proximate (evoking) cause of amyotrophic lateral sclerosis. If previously weakened cells and tracts exist in the spinal-cord, severe concussion or over-innervation (strain) can become the last straw that shatters the structure. Possibly congenital defect will some day be shown to be the ultimate cause (*causa sine qua non*) predisposing to the breakdown.

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"FIBROSITIS" AS A CAUSE OF CHRONIC HYPERTROPHIC SPONDYLITIS, AND POSSIBLY WITH A HERETOFORE UNDESCRIBED SIGN IN INTESTINAL AUTO-INTOXICATION.

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Having studied a considerable number of cases of chronic "rheumatic" myositis [1] and having observed in these cases many instances where the skin, fasciæ, fat and periosteum as well as muscles were involved, and also finding tenderness and stiffness quite commonly present about the spine in many persons in whom other parts were affected, it has seemed to me that chronic hypertrophic spondylitis is often but the spinal localization of a very common general condition, and which, in a mild degree, is quite frequently present. In many instances these various manifestations appear to have a common cause, this being auto-intoxication of intestinal origin. Indeed, such an origin for arthritis deformans has been frequently mentioned by writers, and notably in this country by McCrae,[2] Richardson[3] and Goldthwait,[4] but perhaps heretofore it has been more conservatively expressed.

That this hypertrophic spondylitis existed in the ancients is proven by the adventitious osseous formations found about the spine in skeletons that have been preserved through ages; and that this arthritis is of undoubted frequency in a varying degree is readily proven by the condition being observed in the dissecting-room and by the common appearance of spurs and more extensive osseous formations about the spine as shown by skiagraphs.

Frequently these spurs are so placed as to give rise to no symptoms whatever, but they may interfere with the spinal articulations and at times even impinge upon nerve roots. And depending chiefly upon this nerve root impingement, two types of so-called rhizomelic spondylosis were described. When nerve root symptoms were prominent, the case belonged to the group described by Von Bechterow;[5] if the nerve roots escaped or were but slightly impinged upon, we had the type of Marie-Strumpell,[6] where to the spinal affection was added involvement of some of the major articulations. The difference between these types depended largely upon the site and extent of these adventitious osseous formations. It would seem, then, that, in addition to these two types, many irregular forms might be expected, and this is in accordance with a large number of cases that have been reported. There really are no distinct types of the disease. The feeling of the most careful observers is that the term "chronic hypertrophic arthritis" is the best heading under which to group these various forms.

In an endeavor to maintain my former statement, that I believed hypertrophic spondylitis often to be but part of a very common general condition, I shall go back to the earliest literature obtainable upon this subject. About 1816, Balfour,[7] an English physician, was treating many gouty and rheumatic patients by friction, compression and percussion; he believed the trouble to be chiefly resident in the connective tissue, but I do not know of his having found and described the indurations of which mention is now to be made. It probably was Froriep [8] who first recognized thickenings of this nature in the periosteum, although I am not aware of his having observed any special areas along the spine. Froriep spoke of these thickenings as *rheumatische Schwiele*, and found the condition present in the skin, fasciæ, muscles particularly, and in the periosteum. But few persons appear to know of a highly instructive volume by this German physician, published in Weimar in 1843, and in which he gives most careful clinical records of these "rheumatic" areas in more than eighty cases, telling of their development in many parts of the body. He labored a long time to establish the diagnostic value of the "rheumatic" induration. Froriep said,[9] "To find a new and hitherto undescribed symptom in a complaint of such common occurrence seems nearly impossible; yet in other things how frequently it happens that something is pointed out which has escaped previous observation, and then every one is surprised that he should never have noticed it!" More than half a century has elapsed since then and we are only beginning to recognize its importance, and although for more than fifty years the Swedes have had excellent manual methods for the removal of these areas, it remained for Edinger,[10] the German neurologist, to impress upon the profession outside of the Scandinavian countries the importance of these areas in the production of headache. This physician never failed to acknowledge the debt he owed to the Swedes, from whom he learned of this condition. And, strangely enough, Norström, one of the Swedish physicians whom Edinger mentions, spent some time in this country a few years ago, when he called our attention to the frequency of the condition and very explicitly explained to us the method of treatment. While here, Norström contributed a series of articles on massage to the New York medical journals, and considered the subject more scientifically than any American physician who has yet written upon the manual method of treatment. Unfortunately for us, we have not yet learned the lessons he taught.

For want of a better name, these areas have been termed " rheumatic," but not in the sense that they are the sequelæ of acute inflammatory rheumatism, although occasionally they may be. The term " fibrositis " suggested by Gowers[11] seems to be the best that has yet been offered, though special names indicating the sites of the indurations are useful. In this connection Gowers speaks of " cellulitis " and says, " But we apply the word to those conditions that are hyperplastic in nature and produce induration or suppuration. I think we need a designation for inflammation of the fibrous tissue which has no such results and yet, we cannot doubt, is of this nature. We may conveniently follow the analogy of " cellulitis " and term it " fibrositis."

A new and popular work says of the *induration* as a foci of *chronic myositis,* " But there are, as far as I know, no histological examinations on which to base such a term!" This pathology has been written about in Sweden, Germany, to some extent in England, and it has been discussed, though somewhat superficially, in this country. Briefly stated as typified by involvement of the muscular structure, there is in the beginning a serous exudate, or, in the more severe cases, a serofibrinous exudate; in the chronic areas, an increase in the intermuscular connective tissue and round cell invasion is observed, while the muscular fibers frequently show more or less distortion.

Of course it is important to distinguish between a stiff back due to adventitious osseous formations and that due to chronic muscular rheumatism, and this may be done with the administration of an anesthetic, when the latter condition yields readily, while the former remains unchanged. A skiagraph will also help to clear up the diagnosis.

Somewhat analogous, though resulting from an acute disease, is the frequent development of typhoid spine in cases of typhus abdominalis; from the careful observations of McCrae [12] and others, this spinal condition seems to be largely a spondylitis. A similar condition is frequently present in a mild degree along the cervical vertebrae after attacks of influenza.

The upstart and irregular school of mechanotherapy styled "chiro-practic," which, theoretically at least, differs from osteopathy, claims as a basis for its work a partial occlusion of the intervertebral foramina through displacements of the vertebræ; they make the ridiculously absurd assertion that this is the cause of over 90% of all diseases. It may be that quite often there is present a slight constriction at these foramina, but I feel that this is most probably due to proliferation of the periosteum ("fibrositis") surrounding these openings and giving rise at times to nerve root symptoms. Long,[13] at a necropsy upon a patient who had died of pellagra, actually found this condition present and was unable to pass a Japanese toothpick through foramina which should have admitted a much larger body; radiographs from other patients likewise showed involvement of the spinal foramina. Others have observed the same narrowing of these orifices in spondylitis deformans. The ligaments are sometimes involved and osseous formations have also been found within the vertebral canal.

Many years ago, Geheimerath Oldevig was conducting a private institute at Dresden; his treatment consisted of the manual method and hydrotherapy. He was most painstaking in his work, often spending hours in the physical examination of a patient. For his exceptional skill he was accorded the highest medical honor that was bestowed by the king of Saxony. Oldevig repeatedly called the attention of his pupils to the presence of tender and thickened areas on the lateral aspects of the lumbar vertebræ, in patients who had suffered long from chronic indigestion. Upon palpation, I have found a thickening of the periosteum, with tenderness about the dorso-lumbar spine, in a number of patients who have shown the symptoms of intestinal auto-intoxication. The abdominal symptoms which these cases usually present are constipation, flatulency and often more or less tenderness and rigidity of the abdominal wall; after this rigidity has been overcome by skillfully performed massage, deep pressure will then usually reveal the presence of very tender areas within the abdominal cavity; to me, the exact location of these areas is an exceedingly interesting problem, and the fact that they can often be removed by massage, thereby causing a marked improvement in the general condition of the patient, should be a matter worthy of our most serious consideration.

Undoubtedly the thickened and tender areas upon the vertebræ often undergo absorption, but if they recur, and remain sufficiently long, there may be proliferation of the periosteum, leading to the osseous formations seen in skiagraphs and met with in the cadaver, and which we know are most common in the dorso-lumbar region of the vertebral column. So far as I know, this observation has had no special mention in medical literature, but it is possible that Rose[14] has observed the same local condition in women suffering from backache; he speaks of this as being due to a chronic periostitis of the spinal processes, generally of the lumbar or sacral, and asserts that the affection is easily recognized by pressure with the finger along the spine; he further states that he has had over thirty years' experience with this particular affection, but, except Dr. A. Jacobi, who first called his attention to it, that he knows of no physician who has written or spoken about it. He asks how many women may be treated, often heroically, for uterine trouble, who suffer from this chronic periostitis.

It is interesting to note the association of this subject with dentistry; dentists have long known of the occurrence of pyorrhea alveolaris in persons who were either gouty or rheumatic. In some of these cases osseous formations have appeared upon the teeth; sometimes they are known as calculi, and other forms are scaly in nature. When we recall that the teeth form articulations with the alveolar sockets, and that the roots of the teeth are covered by a modified periosteum made up of fibrous tissue, it is not surprising that a "fibrositis" should be set up in this membrane and at times lead to the development of osseous formations similar to those found along the spine and about other articulations.

The disease, otosclerosis, meaning a progressive middle-ear disease in which the chief pathologic feature is the formation of an osseous deposit, in some subjects may be due to this condition.

It appears that the usual factors concerned in the formation of these areas may be trauma, refrigeration and dampness, and perhaps some poisonous substance elaborated within the body.

Because of the number and variety of movements which the spine is called upon to perform, this part is particularly liable to injury; we know that the lumbar and cervical regions of the spine have the greatest range of mobility, and these are sites where hypertrophic osseous tissue so frequently develops; admitting the presence of some poisonous substance in the circulation, it could readily cause irritation of this injured tissue, and if continued sufficiently long, lead to hypertrophic bony formations. The artificial production of callus by the injection of fibrin beneath the periosteum has recently been reported by Bergel,[15] and a familiar example of the ease with which osseous tissue is produced is recalled in the callus that forms after fracture.

The effect of cold and dampness is such a common experience, as when a cold draft strikes the back of the neck, and the pain and stiffness which follows unfavorable weather conditions, as to need but little mention. It is perhaps in some such sense as this that the word "rheumatic" is most applicable. This effect is by no means limited to the muscles, for the periosteum is frequently involved. I am aware of the incredulous attitude of some physicians towards such effects from refrigeration; but how illogical for such an one to admit that he has not even recognized the condition of indurative headache, "perhaps the most common form of headache."

As to the presence within the body of some circulating poisonous substance, I recall a number of instances in which indurations in the skin, fasciæ and muscles were being broken up by vigorous massage, where the patient failed to keep up good elimination through the bowel, kidneys and skin, that headaches were felt, and neuralgic and rheumatic pains complained of in different parts. In one case which I have referred to elsewhere,[16] a typical attack of gout in the metatarso-phalangeal articulation of the great toe appeared to be precipitated in this way; the pain, redness and swelling, which developed so suddenly, had never been experienced before, nor has it been followed by any similar attack. It is true that no such poisonous substance has yet been isolated, but it may be similar in nature to some of the toxic matter which we believe so often slowly produces general arteriosclerosis. These cases frequently have arteriosclerosis before old age overtakes them, and the change in the vessel walls in the first stage of arteriosclerosis is similar to that so often observed in the chronic indurations met with in the connective tissue in many parts of the body.

Another possible cause, and one worthy of most careful consideration, is micro-organisms; and it may be that, later, various organisms will be shown to be directly responsible for the development of some of these indurations.

These various causes may or may not all be responsible for the production of hypertrophic osseous tissue present about the spine; but that a thickening exists, and that tenderness and sometimes pain can be elicited by pressure with the fingers over these areas, there is no doubt; and, finally, if these areas are not absorbed, I believe they often lead to the adventitious osseous tissue seen in skiagraphs and met with at necropsy.

The study of this low-grade inflammation in the connective tissue is big with possibilities. Its importance in the muscular system alone may be realized when a recent writer[17] considers it "an every-day affection" and says, "So much for a brief historical survey of what I consider to be the most neglected subject in the English literature of to-day."

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[10] Norström: Chronic Headache and Its Treatment by Massage. New York Med. Jour., Nov. 4, 1905, vol. lxxxii, no. 19, p. 956. Yawger: Indurative Headache (Schwielenkopfschmerz), with Report of Three Cases. Jour. Am. Med. Asso., April 24, 1909, vol. lvi, p. 1316.

[11] Lumbago: Its Lessons and Analogies. Brit. Med. Jour., Jan. 16, 1904, no. 2246, p. 117.

[12] Bulletin Johns Hopkins Hosp., March, 1911, vol. xxii, no. 240, p. 75.

[13] Pellagra. Jour. Am. Med. Asso., Aug. 27, 1910, vol. lv, no. 9. p. 734.

[14] The Cause and Treatment of Backache in Women. Jour. Am. Med. Asso., Jan. 23, 1909, vol. lii, p. 314.

[15] Begel: Callusbildung durch Fibrin. Arch. für klin. Chir., Bd. 93, Heft 3, p. 775.

[16] Osteopathy: Does It Offer Anything New? Therap. Gaz., July 1910, vol. xxvi, p. 436.

[17] Telling: "Nodular" Fibromyositis, an Every-Day Affection. Lancet, London, Jan. 21, 1911, vol clxxx, p. 154.

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AMYOTONIA CONGENITA: A CLINICAL AND PATHOLOGICAL STUDY.

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The disease called by Oppenheim[1] "myatonia congenita," but for which the term "amyotonia congenita" is rather to be preferred, as less liable to cause confusion, was first described only eleven years ago. Since that time it has continued to arouse much interest, although its nature and true position among diseases are still not well understood and the number of reported cases is not great.

It consists, in brief, as originally described by Oppenheim, of a flaccid paralysis, apparently congenital, most marked in the extremities, especially the lower, and varying in degree up to almost complete paralysis of the whole body. There is no actual atrophy visible; the sensation is not disturbed; the tendon reflexes are diminished or abolished; the electrical contractility is much diminished; the hypotonia is so great that there is unusual passive mobility at the joints, and in bad cases the child is unable to sit and the head falls loosely in any direction.

One of us (Griffith[2]), in reporting, in May, 1910, a new case, that of Charles K., was able to collect and detail in abstract form but 48 other instances which seemed properly to belong in this category. In this article a study of the symptoms and pathological findings in the reported cases was also made and a bibliography appended. Some years earlier one of us (Spiller[3]) had published a personal case with the post-mortem findings. This was the first case reported in this country, and the first with necropsy reported by anyone.

Since the publication of the history of Charles K., above referred to, the child died, and we were fortunate in securing an autopsy.

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[1] Monatsschr. f. Psychiat. u. Neurol., 1900, viii, 232.

[2] Archiv f. Kinderheilk., 1910, liv, 241; also Trans. Amer. Ped. Soc., 1910.

[3] Univ. Penna. Med. Bull. 1905, xvii, 342.

This is the second case with autopsy occurring in America. It is our purpose in the present paper to analyze briefly the clinical data given in the reports of cases published since the writing of this article, so far as known to us, and to discuss the pathology of the disease in the light of the findings in our own cases and in those of others.

Among the cases to be considered here we include those of Ashby and of Moussous and Carles, referred to in a footnote, but not analyzed in the earlier publication.[4] In giving these and other abstracts we have pursued the method employed in this publication, numbering them in continued series with those there detailed, and discussing symptoms briefly; this clinical portion being by way of an appendix to our earlier study of 49 cases (Griffith).

Case L.—Ashby.[5] Girl, aged eight years; family history negative; dated from birth. In bed, almost completely paralyzed; all muscles extremely flabby and wasted, including those of the face, which has little expression; can barely move limbs; cannot sit; some difficulty in mastication; reflexes absent or feeble; electrical reactions normal; intelligence somewhat impaired. Improved slightly under treatment.

This case is in many respects anomalous, especially in the extreme wasting and the unimpaired electrical reactions.

Case LI.—Moussous and Carles.[6] Girl, aged four years and three months; family history negative; dated from birth. Cannot walk; can move arms slightly and feebly; cannot sit erect or roll over; face seems a little inert; no apparent atrophy but muscles soft; electrical contractility lessened; no reaction of degeneration; intelligence a little diminished.

Case LII.—Naish.[7] Boy, aged four years and eleven months; family history negative; dated from birth.

Examination. Hypotonia of all muscles of the trunk and limbs, but especially of the upper limbs; sits, with kyphosis; stands and walks uncertainly; all movements weak; knee-jerk almost absent; electrical reactions diminished; great passive hypermobility; intelligence normal.

Case LIII.—Openshaw.[8] Boy, aged seven years and six months; could move arms and legs until measles at eighteen months, since then no power in legs; never walked; power in arms diminished slightly, but can write legibly; sits, with lordosis; cannot hold head up; decided wasting of muscles in arms, nothing said about legs; knee-jerk absent; contracture at hips, knees, and ankles; passive hypermobility of elbows; intelligence normal. "The muscles of the legs reacted to faradaic, and gave a brisk contraction with the constant current." Improved decidedly under treatment.

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[4] Griffith, loc cit.

[5] Arch. of Pediat., 1910, xxviii, 363.

[6] Arch. de Méd. des Enf., 1910, xiii, 472. From Jour. de Méd. de Bordeaux, 1909, No. 44, 697.

[7] Proc. Royal Soc. Med., 1909-10, iii, Neurological Section, 95.

[8] Ibid., Clinical Section, 39.

Biopsy of a portion of hamstring muscle showed "fatty infiltration; absence of cross-striation; irregular clefts with granular debris, with signs of regeneration in various parts."

The history as given leaves open some element of doubt regarding the diagnosis. It is not clearly stated whether the condition was congenital. The apparently sudden development, or rapid increase, of loss of power in the legs following measles, and the wasting in the arms are also not in accord with the usual picture of amyotonia congenita.

Case LIV.—Zahorsky.[9] Boy, aged two years and three months; could not walk; knee-jerk diminished; no electrical contractility; "everything very flaccid." Improved somewhat.

The author designates this a case of myatonia, but the very meagre details given in the brief society report leave the important diagnostic symptoms unstated. Possibly a fuller account will appear elsewhere.

Case LV.—Skoog.[10] Girl, aged twenty-two months; family history negative; could sit and support head at six months; never crawled or attempted to walk. Disease not noticed by parents until fourteenth month.

Examination. Partial flaccid paralysis of arms and legs; all movements weak; cannot walk except a few steps when supported; holds head erect; sits, without kyphosis; possibly slight weakness in muscles of facial nerve; excessive hypermobility on passive movement; well nourished; muscles soft; tendon reflexes abolished; feeble galvanic and faradic response in some muscles, not in others; no reaction of degeneration.

Microscopic Examination of Muscle. Small piece of gastrocnemius removed. A section from immediately beneath the muscle sheath contained no muscle bundles, all being substituted by fatty tissue. A deeper section contained some small, pale muscular tissue and much fat; "some muscle fibers in process of destruction, and seem ending in continuity with substituted connective tissue;" much fatty tissue between bundles; no hypertrophied fibers; all quite uniformly atrophied; great nuclear proliferation. Blood-vessels have greatly thickened walls.

The history of this case leaves some doubt about its exact nature. Strongly against the diagnosis of congenital amyotonia is the absence of a positive history of congenital origin. This makes it seem possible that the disordered condition developed later and had been an advancing one. On the other hand, more careful observation by the parents might have thrown a different light on the case.

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[9] Amer. Jour. Obstet., 1910, lxii, 1132.

[10] Jour. Amer. Med. Assoc., 1910, lv, 364.

Case LVI.—Hummell.[11] Boy, aged three years; family history negative; dated from birth; never crawled or walked. Cannot stand; sits, with kyphosis; head falls forward when fatigued; moves legs, although feebly; upper extremities involved, but less so; decided passive hypermobility; muscles soft; tendon reflexes abolished; slight diminution of galvanic and great diminution of faradic contractility; no reaction of degeneration; no contracture. Ten months later there was decided improvement and he could stand with assistance.

Case LVII.—Chéné.[12] Girl, aged six months; family history negative; fetal movements felt; paralysis noticed by mother at end of second month, but disease probably congenital.

Examination. General flaccid paralysis; head falls any direction; kyphosis; cranial nerve supply normal; slight movements of hands and feet; limbs of normal size, but muscles feel soft, and fatty cutaneous tissue prevents determination of actual atrophy; extreme passive hypermobility of joints; knee-jerk and ankle-jerk absent; sensation and intellect normal; faradic contractility much diminished; galvanic response only with ten milliampères.

Case LVIII.—Chéné,[13] second case (unpublished case under care of Comby). Boy, aged ten months; family history negative; dated from birth. General feebleness of musculature; head supported with difficulty; moves limbs while lying in bed but ceases when lifted up; has difficulty in keeping himself erect when held on nurse's arm; muscles soft, no positive atrophy; no paralysis or contracture; knee-jerk diminished; passive hypermobility of ankles. Very large fontanelle and thorax flattened at the sides; rachitic rosary. In two months improved very greatly; could stand holding to objects; could hold objects in hands; knee-jerk present.

This case was carefully observed by a physician (Comby) with experience in this disease, and is doubtless a genuine one. The report, however, does not make it clear that decided, early rickets was not a factor of importance.

Chéné, in his list of cases, included one reported by Gayarre,[14] of which we have not seen the original report, but from the brief extract given the case hardly appears to belong here, inasmuch as exaggerated reflexes and clonus were prominent symptoms.

Case LIX.—Pollak.[15] Boy, aged six months, family history negative; fetal movements felt; dated from birth; arms and legs affected at first, but arms have improved somewhat.

Examination. Head falls to side as in newborn; marked kyphosis; respiration entirely abdominal; tone of abdominal muscles diminished; upper and lower extremities show flaccid paralysis; arms and toes move slightly on strong stimulation; muscles soft, not surely atrophied, as fat covering is well developed and prevents examination; movements of muscles supplied by facial nerves decidedly diminished, as seen when child cries; sucks with little power; muscles supplied by hypoglossal nerves much involved, and tongue can be depressed by finger with great ease; great passive hypermobility of joints; tendon reflexes absent; no electrical reaction of muscles or nerves obtained; sensation normal. Improved decidedly in course of a month, but died three months later of "capillary bronchitis." Thymus found enlarged. Later report of autopsy to follow.

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[11] Jour. Nerv. and Ment. Dis., 1910, xxxviii, 749.

[12] Thèse de Paris, 1910.

[13] Ibid.

[14] Revista Clin. de Madrid, June 15, 1909.

[15] Archiv f. Kinderheilk., 1910, liii, 373.

Case LX.—Variot and Chatelin.[16] Girl, aged three and one-half years; family history negative; symptoms noted at six months; said to have been worse at two years than when reported.

Examination. Can make different movements with legs, but feebly; upper extremities involved but to less extent; sits, with kyphosis; cannot stand; holds head erect; passive hypermobility; thoracic movements normal; muscles soft without evident atrophy; tendon reflexes abolished; slight contracture at hip; faradic contractility almost abolished, galvanic much diminished; slight suspicion of degenerative reaction, but probably not genuine; intelligence normal.

The almost certain congenital origin and the decided improvement make this a well-marked case, the positive reaction of degeneration not being certain enough to become an element of doubt.

Eleven additional cases, of which the abstracts have been here given, possibly belonging to the category of amyotonia congenita, have, therefore, come to our notice since the report of Charles K., and the analysis of other cases reported up to that date. This does not include two cases referred to by Cassirer,[17] of which we have no details. The cases vary in severity, and agree in the main with the general symptom complex of the disease. Inheritance of any family predisposition was not mentioned in any of them. In all, the disease began at an early age, the symptoms dating clearly from birth in the majority, and probably so in Chéné's first case. In Skoog's patient the symptoms were first noticed at fourteen months, and in Variot and Chatelin's not until six months. This may be through failure of careful observation on the part of the parents. In the cases of Openshaw and of Zahorsky the reports do not make the matter clear. In Openshaw's case the occurrence of an acute disease (measles) certainly made the weakness worse at eighteen months, if it had existed previously, while in Chéné's second case the process of recovery from the well-marked rachitis may have some bearing, to an extent not clear, upon the unusual and rapid improvement in the symptoms exhibited.

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[16] Rev. Neurologique, 1911, xix, 138.

[17] Handb. der Neurol., Lewandowsky, 1911, ii.

Muscular hypotonia was present in the lower extremities in all the 11 cases. Walking unaided was impossible except in Naish's case. In some cases, of course, the early age of the patient at the time the report was made precluded the possiblilty of walking in any event. The arms were more or less involved in all cases, but generally less than the lower extremities, as far as data are given on this point. Only in the case of Naish was the trouble greater in the upper extremities. In Oppenshaw's patient there appeared to have been very little affection of the arms.

When sitting was possible, as it was in about half the cases, spinal curvature was liable to appear. The face is reported involved in the cases of Ashby (little expression), Moussous and Carles (a little inert), Skoog (possibly slight weakness), and Pollak (decided diminution of motion, with involvement of the hypoglossal nerve). This is an unusual proportion in comparison with that generally reported, involvement of the cranial nerves being uncommon. Interference with thoracic respiration is mentioned by Pollak only. Decided hypermobility on passive movement is referred to in 9 cases. In the other 2 cases (Ashby and Zahorsky) no reference is made to the matter. No certain wasting of muscles was discovered, except in the cases reported by Openshaw and Ashby, but it is often stated that although the limbs seemed properly rounded, they were evidently soft, and the presence of the fat layer prevented the existence of atrophy being determined. The patellar reflexes were absent or greatly diminished in all cases except that of Moussous and Carles' patient. The original of their publication was not accessible to us, but at least no reference was made to it in the abstract. The electrical reactions were diminished in 8 of the 11 cases. In Chéné's second patient no reference is made to them; in Ashby's patient they were found normal, this case being anomalous in that as in other respects; and in Openshaw's case there would appear to have been no diminution, but the matter is not quite clear. A suspicion of a degenerative reaction was found in Variot and Chatelin's patient, but the condition was considered not a genuine instance of it. Some diminution of intelligence was reported by Ashby and by Moussous and Carles, but was evidently not of moment, and must be considered accidental, or apparent only. Contracture was present in the cases of Openshaw and of Variot and Chatelin. More or less improvement is said to have taken place in 7 instances. In Ashby's and Zahorsky's patients it was slight; in Openshaw's, Hummell's, Pollak's, and Variot and Chatelin's it was decided; in Chéné's second patient it was so great that it appeared to be largely due to improvement in the rachitis present. Only 1 death was reported (that of Pollak's patient) due to respiratory involvement, as has so often been the case in this disease. This was the cause of the fatal ending in our patient, Charles K.

The history of Charles K. up to the last notes made at the time of reporting is, in abstract, as follows:

Boy, aged fifteen months; admitted to the Hospital of the University of Pennsylvania, December 8, 1909. Parents, drunkards. Child had general flaccid paralysis from birth, and had never been able to use his legs or handle objects. Examination showed inability to sit or (Figs. 1 and 2) to hold his head erect; only very slight movement of arms and legs possible; diminished expansion of the thorax; no rickets, no wasting of the limbs; knee-jerk absent; moderate passive hypermobility of joints; slight contracture; entire failure of faradic and galvanic response. Under treatment up to April 26, 1910, the child improved considerably.

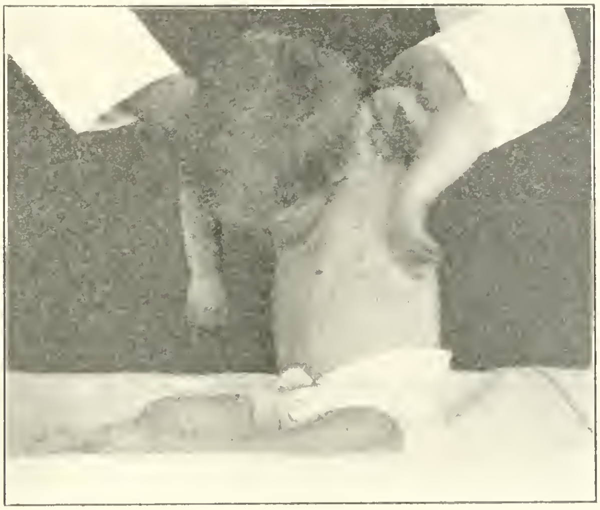

Fig. 1.—Photograph of Charles K.

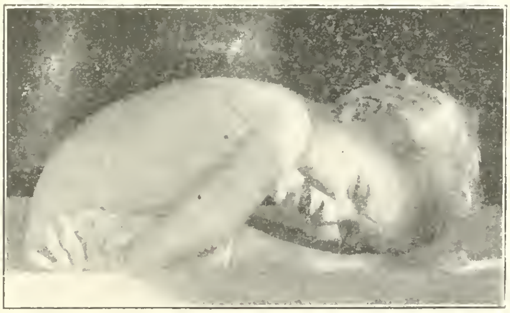

Fig. 2.—Photograph of Charles K.

The later history of the child reads: During the month of May, 1910, the boy had more or less fever; there was decided improvement in the ability to hold the head erect, but he could sit only when propped with pillows. The abdominal reflexes were absent; respiration was still almost entirely abdominal; the slight contracture previously mentioned persisted. Anemia was decided, and this, with irregular temperature, continued until July. He was then sent to the seashore, but returned in September in about the same condition as when he left the hospital Little change of any sort took place, and there was certainly no improvement from this time. About the end of October, 1910, he developed bronchitis, with fever, then pneumonia, and died after an illness of five days.

The autopsy was performed by Dr. B. S. Veeder, and his report and our findings in the nervous and muscular tissues are the basis for the statements which follow:

General. Body of a white male infant, about two years of age. The body was well nourished; no emaciation present; postmortem rigidity absent; practically no lividity; no scars or external markings. The skin was very pale, soft, and delicate in texture; no enlargement of the superficial lymphatics present; fontanelles closed. The autopsy was performed at a private house, under circumstances which made several features rather unsatisfactory.

Incisions were made in order to obtain nervous and muscular tissue. On incision of the calf the subcutaneous fat was found markedly increased, and measured 1.5 cm. in thickness; the bloodvessels showed as pale white cords; the muscle tissue markedly atrophic, being rather tendinous in character, of a very pale color, and soft and flabby in consistency. A similar condition was found in the forearm, where the muscle surrounding the bone was simply a thin layer about one-eighth inch in thickness, the rest of the arm, which was o about normal size for the age of the child, being made up of adipose tissue.

Abdomen. On opening the abdominal cavity the fascia and subcutaneous tissue were found thickly infiltrated with a large amount of adipose tissue; the abdominal muscles little more than a thin sheath of tissue; the intestines distended, pale white, glistening in color, without adhesions; the liver extended slightly below the costal margin; no fluid present in the abdominal cavity. The abdominal wall showed a slight greenish discoloration of putrefactive change.

The spleen was moderately firm in consistency; maintained its shape when laid upon a flat surface; weight, 26 grams; sharp edges, rather pale red in color, smooth capsule, measured 7.5 x 4 x 2.5 cm. The cut surface was rather pale, not very moist, bled slightly, and showed faint markings of trabeculæ and follicles.

The liver was of firm consistency, with sharp edges, smooth capsule, of pale reddish-brown color, weighed 460 grams, and measured 18 x 11 x 5 cm.

The pancreas was dark purple in color, of normal consistency, and 8.5 cm. in length.

The stomach was decidedly distended, the mucosa thin, rather atrophic, grayish in color; covered with considerable mucus, and without ulceration.

A small portion of the ileum was removed for microscopic study, but only the duodenum was examined in its extent. This showed a thin wall, with the mucous membrane slightly grayish in color and without ulceration.

The right kidney weighed 70 grams and measured 10 x 4 x 3 cm.; was firm, rather bulging, and cut with normal resistency. The cut surface was smooth, and showed slight thickening and overgrowth of the cortical portion, which was pale white, and stood out in marked contrast to the dark red, congested pyramids. Throughout the kidney there were numerous small, grayish-white points, particularly in the pyramids and in the pelvic portion. The glomeruli were not visible. The vessels of the cortex showed distinct striation.

The adrenals were small, pale, and distinctly yellow in color.

The mesenteric lymph nodes were about the size of split peas; those of the retroperitoneum were not enlarged.

Thorax. Both pleural cavities were free from adhesions, but a few adhesions bound together the lobes of the right lung. The pericardial sac was free from adhesions or fluid. The lower lobe and almost the entire upper lobe of the left lung were moist, dark blue in color, and firm; the rest of the lung exceedingly pale and crepitant throughout. The pleural surface showed markings of the alveolæ. Section of the dense firm area showed a deep blue, almost cyanotic color; the surface moist and exuding on pressure small white plaques of purulent material, with very slight bleeding. Section of the pale, soft, crepitant areas showed a rather normal appearance, except for quite marked anemia. The right lung weighed 180 grams; both the upper and lower lobes were involved in a bronchopneumonic process similar to that described in the other lung. The middle lobe was uninvolved.

The heart weighed 80 grams. The small, white, firmly contracted chambers contained small, firm, currant-jelly clots attached to the papillary muscles. The right ventricle contained soft clots in addition. The valves and orifices were normal in appearance. The musculature of the heart was rather pale, yellowish gray in color, and of fairly good consistency. The papillary muscles, especially on the right side, were pale and short. The wall of the left ventricle measured 9 mm., and that of the right ventricle 3 mm. in thickness.

The aorta and bloodvessels exhibited no lesions.

The thymus gland was rather small, and extended downward beneath the sternum as far as the second interspace.

The thyroid gland was exceedingly small.

Nervous System. No excess of fluid was found within the skull. The brain substance was soft and the membranes somewhat congested.

The spinal cord exhibited a rather edematous appearance of the membranes, with some coagulated blood extending almost the entire length of the canal.

The following tissues were prepared for histological examination: heart, lungs, liver, pancreas, kidney, adrenal, small intestine, thymus, thyroid, voluntary muscle, brain, cord, and nerves.

Heart: The cut surface showed a normal pericardium and endocardium. The muscle-fibers lay closely together, and both longitudinal and cross-striation were distinct. There was a slight granular condition of the protoplasm present. The nuclei were regular and oval in shape, and with considerable polar pigment. The bloodvessels throughout were filled with erythrocytes.

Lungs: Sections taken from the non-consolidated portion showed a moderate amount of congestion of the alveolar walls with occasional areas of edematous material partially filling the alveolar spaces. Sections from the consolidated part of the lung showed large areas in which the alveolar spaces were filled completely with an exudate consisting of leukocytes and broken-down cellular material. In most of these areas several bronchi could be made out in which there was almost complete desquamation of the epithelium, and the lumen filled with broken-down and disintegrated cells, and with leukocytes. Occasionally areas were seen in which the alveolar spaces were only partially filled and in which some air was still present. There was marked congestion throughout.

Liver: The capsule was normal, and there was no increase in the perilobular connective tissue. The central veins were empty, and only a few capillaries were injected. Throughout the entire lobule the liver cells contained large vacuoles, occasionally forming quite typical seal-ring cells. The lumen of the larger bile ducts was filled with faintly staining granular material.

Pancreas: The interstitial and periductal connective tissue was not increased in amount. The cells of the acini stained well and did not show degeneration. The islands of Langerhans were numerous and large and showed nothing abnormal.

Kidney: The capsule was thin and regular. There was no increase in interstitial tissue in any part of the kidney. The cells lining the convoluted tubules stained poorly, and appeared as if smeared with eosin. The protoplasm was slightly granular and cell outline very indistinct. The nuclei were small and regular, and were rather solid, and there had been no loss of nuclei. The free margins of the cells were slightly irregular, and encroached somewhat upon the lumen of the tubules. The capillaries between the tubules contained few erythrocytes. The glomeruli were large, and almost completely filled the spaces. There was no exudate in the spaces, nor were there any evidences of proliferation present. The nuclei of the tufts were numerous and densely packed together, as is usually found in a young kidney. The bloodvessels of the tufts were well injected. Throughout the section marked congestion was seen.

All of the changes were explainable by a condition of postmortem degeneration, but as the other organs did not show this to such a degree, it is possible that there had been a cloudy swelling present.

Adrenal: The capsule and interstitial tissue showed no abnormality. In the cortex the three zones were distinct. The medullary portion was compact, and contained several large bloodvessels with a clear lumen. The nuclei of the cortical cells were large and somewhat vesicular, and stained deeply. The protoplasm was highly granular and stained rather faintly, but the large vesicles so frequently seen were not present. The only noteworthy feature of the medul ary portion was the absence of cells bearing chromatin pigment.

Small Intestine: The mucosa of the duodenum was in a decided state of postmortem degeneration. The submucosa showed marked hyperplasia of Brunner's glands, which in places composed over half the entire thickness of the intestinal wall. The cells of the glands were granular and somewhat disintegrated. The other layers showed nothing abnormal. The ileum showed likewise considerable postmortem degeneration of the mucosa, without other changes of note in the section.

Thymus: The thymus had undergone but little involution, the fibrous tissue between the lobules not being marked. The lobules were densely packed with cells. These were chiefly small, deeply staining lymphocytes, but numerous large cells, with oval and vesicular nuclei, were present. The corpuscles of Hassal were not very numerous, and were not fully developed. In many places there were small collections of the larger cells noted in which the cells were beginning to group themselves in a concentric manner, and transitions from this stage to a quite typical Hassal's corpuscle could be distinguished. Numerous eosinophilic cells were present, and these showed some slight tendency to occur in groups. There was a marked congestion of the bloodvessels.

Thyroid: The thyroid showed nothing distinctly abnormal. In a few places there was a slight hyperplasia of the epithelial cells lining the acini, and although the acini were uniformly filled with considerable colloid material, there was no dilatation of them.

The gross anatomical diagnosis consisted then of atrophy of the muscles, with general fatty infiltration; bronchopneumonia; cloudy swelling and congestion of the kidneys, and slight cloudy swelling of the liver. The histological diagnosis, not including the muscular and nervous system, was bronchopneumonia, cloudy swelling of the kidney, and fatty infiltration of the liver.

As special interest attached to the condition of the muscular and nervous systems, we have given particular attention to this part of the pathological findings, with the following results:

The brain was unusually large, and appeared more like the brain of an adult than that of a child. It weighed 1542 grams after it had been one week in a 10 per cent. formalin solution. The cord measured 23 cm. in length, and as it had been removed close to the foramen magnum, the difference in the relative size of the brain and cord was striking. The posterior spinal roots appeared to be of good size, and a transverse section of the cord was not unusually small. The anterior roots throughout the cord were small.

The cells of Betz in the right and left paracentral lobules were numerous, especially in the left, although no significance is to be attributed to this apparent excess on the left side. In some of the Betz cells the chromophilic elements were in normal form; in others they had become granular. The cells of the hypoglossus nuclei may have been a little diminished in number, but there was no noteworthy atrophy comparable with that of the cells of the anterior horns of the spinal cord.

Sections from the seventh and eighth cervical segments showed few nerve cells in the anterior horns, except that in the eighth cervical segment a group of cells was present on each side corresponding to the intermediolateral tract. The few cells remaining in the anterior horns were small, but contained chromophilic elements. No degeneration in the white matter was detected by the Weigert hematoxylin stain. The anterior horns by this stain appeared a little pale, as from a scarcity of nerve fibers.

The seventh and eight posterior cervical roots, cut separately from the cord, were in excellent condition, showing no degeneration. The anterior roots from these levels were degenerated, but somewhat less so than the anterior roots of the lumbar region. No evidence of degeneration by the Marchi stain could be found in sections from the cervical, thoracic, or lumbar region.

The anterior horns of the lumbar region were paler than the posterior horns. The nerve cells of the anterior horns were few, and most of those present were shrivelled. The cells of Clarke's columns were in good condition; some showed alteration—namely, chromatolysis and peripheral displacement of the nucleus—but such findings are not unusual in the cells of these columns.

The nerve cells of the anterior horns in the lumbar region did not show any noteworthy change by the Bielschowsky method. The first, second, and third anterior lumbar roots, cut separately from the cord and stained by the Weigert hematoxylin method, showed great diminution in the number of medullated fibers, and a considerable overgrowth of connective tissue was evident when acid fuchsin and hemalum were used for staining.

The first, second, and third posterior lumbar roots appeared to be normal. The difference between the anterior and the posterior lumbar roots was very striking, as the former were much degenerated.

Portions of muscle were taken from the back near the neck, from the forearm just below the elbow, and from the calf. The muscle from the forearm showed great atrophy of muscle fibers, but even these greatly atrophied fibers preserved their transverse striation, although longitudinal striation was not distinct. The atrophy affected certain bundles, and other bundles in the immediate vicinity in large measure escaped. The intramuscular nerve fibers stained by the Weigert hematoxylin method showed considerable degeneration, and in some nerve fibers the medullary substance appeared as broken into small black balls. Here and there in transverse section a bundle of rounded, unusually large muscle fibers was found. The atrophied fibers showed an excessive number of sarcolemma nuclei, in some places the nuclei forming dense masses, and almost obscuring the greatly atrophied muscle fibers. By careful examination the atrophied muscle fibers usually could be detected. The interstitial tissue was considerably increased. Some of the muscle fibers showed fatty changes within them by the Marchi method, in the form of minute droplets in small points.

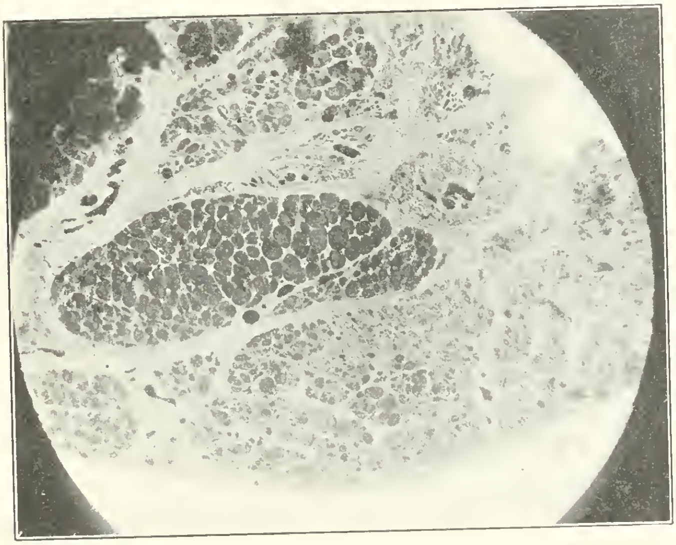

FIG. 3.—Section of a muscle from the back, showing the atrophy of many muscle fibers with a bundle of nearly normal fibers in the midst of greatly atrophied fibers.

Muscle from the back resembled that from the forearm, but was in better preservation; that is, the atrophy was not so intense. There were many highly congested bloodvessels. Marchi's method revealed fatty degeneration within the muscle fibers, but not so much as in the muscle from the forearm. Intramuscular nerve fibers stained by the Weigert hematoxylin method appeared partially degenerated. Some large muscle fibers were found. Many muscle fibers appeared rounded in transverse section. Some muscle bundles were little affected, and yet were surrounded by bundles of greatly degenerated muscle fibers.

The calf muscle was similar to that from the forearm, but the changes were more intense in the former. The muscle fibers were more atrophied, and the interstitial fibrous tissue was much increased in amount. The connective tissue nuclei were distinctly more numerous. Fatty change was evident by the Marchi method within many of the muscle fibers. There was an excessive amount of fat between them. A piece of nerve, probably the posterior tibial, cut with the muscle, showed much degeneration by the Weigert hematoxylin method. Intramuscular nerve fibers were considerably degenerated. The muscle fibers within the muscle spindles appeared to be normal. The bloodvessels within the calf muscle were much sclerosed.

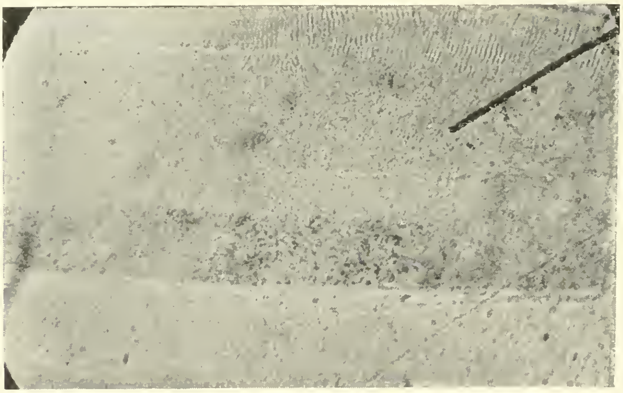

Fig. 4.—Section of muscle from the calf stained by the Marchi method, showing fatty change within a muscle fiber.

A piece of the median nerve teased and stained with 1 per cent. osmic acid solution showed no pronounced degeneration by this method.

A summary of the important findings in the muscular and nervous systems in this case is as follows:

The brain was of extraordinarily large size for so young a child (twenty-five months), and was as large as that of an adult. Excepting the large size, it presented no gross anomaly. The nerve cells of the anterior horns in the cervical and lumbar regions were scarce, and those present were much atrophied. The anterior roots throughout the cord were considerably smaller than in a normal spinal cord, and stained imperfectly by the Weigert hematoxylin method. A peripheral nerve examined was much degenerated. The alteration of the muscles was intense, and consisted of greatly atrophied muscle fibers in the midst of other fibers well preserved, of overgrowth of sarcolemma nuclei and connective-tissue nuclei, of excess of fibrous and fatty connective tissues, of increase in size of some muscle fibers, and of fatty change within certain muscle fibers. The intramuscular nerve fibers showed degeneration. The muscle spindles appeared to be normal.

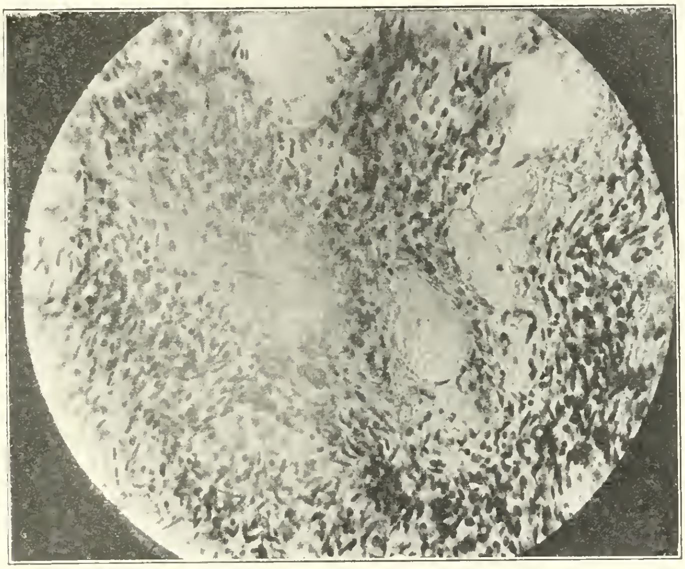

FIG. 5.—Section of muscle from the calf, showing the great excess of sarcolemma nuclei and connective-tissue nuclei and of fatty tissue.

A comparison of this case and of its pathological findings with the case B. T., previously reported by one of us (Spiller), is instructive. The condition in B. T. corresponded closely with the description of the disease as given by Oppenheim. Objections have been made to it by one or two authors on two points—namely, a so-called blindness and a so-called paralysis of deglutition. Neither of these conditions was recorded in the history. The case has been accepted as typical by many, especially by such important investigators as Collier and Holmes, and Cassirer.

The baby was born at full term, and the birth was normal. It was breast-fed until the date of its admission to the hospital. Nothing abnormal in the child was noticed by the parents until he was about five months of age, when he did not seem to take notice of things as he should. He had never been able to hold anything in his hands. The mother had not noticed any increase in the child's weakness since birth, and as he was very weak when he came under the observation of one of us, it is reasonable to suppose the child was equally weak at the age of five months. The mother was an intelligent woman, and her statements were reliable. It is presumable that so marked a weakness might readily escape detection by the parents in the first months of life, and it is equally presumable that a child so weak would not notice objects in a normal manner. So far as the ocular examination can be relied upon it was negative, as shown by Dr. de Schweinitz's explanatory letter accompanying the published report.

The statement made in the report of the case might lead one to conclude that paralysis of deglutition existed, but probably such was not the case. The child was breast-fed until the time of his admission to the hospital, and had shown no difficulty in swallowing. When he was given cow's milk in an attempt to wean him he spit it out; in the report the words are, "And of late, since he has been taken from the breast, has swallowed with some difficulty."

It was not supposed that these words would be taken to mean a paralysis of deglutition. The child had swallowed well while on the breast. Inasmuch as he was twenty-two months of age, and possibly was not receiving sufficient nourishment, and also because it has been suggested that the development of amaurotic family idiocy may be dependent on the mother's milk (a view hardly acceptable), it seemed advisable to endeavor to have the child nourished by cow's milk. That the child would not immediately swallow this, and later swallowed it unwillingly and apparently with difficulty, in no way proves a paralysis of deglutition, and probably such paralysis did not exist. Even if it had, it would not have weakened the diagnosis in any way. As the hypoglossus nucleus has been found affected in amyotonia congenita, it is not unreasonable to suppose that the nuclei governing deglutition some day may be found implicated.

As for the objection to the case based on the existence of a squint, that has already been answered in an article published several years ago.[18] The muscular condition was typically that of amyotonia congenita, and yet the degree of weakness was not so intense as in some of the reported cases.

The pathological findings in this case are important, and the examination was complete. If more findings were not recorded, it was because they did not exist, with the exception of some alteration of the muscle from the left foot, not observed in the sections made at the time of the report. Since that time new sections have been made from all the muscles obtained, but only in those from the foot were additional findings observed. The pathological findings are recorded here in order to compare them with the findings in the case of Charles K.

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[18] Spiller, Neurologisches Centralblatt, 1907, No. 11.

"The spinal cord and brain were well developed, and the anterior and posterior spinal roots were normal. The nerve cells of the anterior horns of the cervical and lumbar regions were normal by the theonin stain. The white matter of the spinal cord was not affected, even by the Marchi stain. A nerve from the upper part of the right upper limb stained by the Weigert hematoxylin or acid fuchsin method was normal. The internal popliteal nerve and a nerve from the upper part of the right upper limb teased and stained by a 1 per cent. osmic acid solution were normal.

"Muscles from the sole of the left foot, from the back of the trunk, and from the left calf had a hyaloid appearance, and that from the sole of the foot was striking, on account of the increase in the nuclei of its connective tissue. The muscle fibres were small, and those from the sole of the foot and from the left calf were much smaller than those from the back of the trunk, and the child had had more power in the muscles of the back than in those of the lower limbs. Unusually large muscle fibers were not observed. The transverse striations were well preserved, but the longitudinal were not so distinct. Nerve fibers within the muscles from the sole of the foot appeared to be normal."

It is difficult to know in what way the report of the muscular condition could have been enlarged upon.

It was not asserted in the report of the case of B. T. that amyotonia congenita must be considered as a purely muscular disease. The statement was, "Congenital myatonia seems from the study of this case to be a muscular disease." It might have been well to italicize the words "this case," but it was not supposed that the statement would be taken to imply that one case could determine the pathology in all its details of a new disease.

The case of Charles K., reported in this present paper, was one of a more advanced type than that of B. T., and the findings were much more pronounced. In the former the nervous system, as well as the muscular, was greatly affected. In recently made sections from the left foot of B. T. some areas were found in which certain of the muscular fibers in bundles were more degenerated than any seen in previous sections, and these were in the midst of fibers of normal size. Some of the muscular fibers had disappeared in these areas. These findings were considerably less intense than those of the muscles in Charles K., and seem to present a transitional stage to the intense muscular degeneration of the latter case.

It is instructive to compare the findings in the reported cases. Beginning with the earliest, Spiller's case, the findings were purely muscular, and not intense although pronounced.

In Baudouin's case the changes in the nervous system appear to have been slight. In the lowest part of the eighth cervical segment the cells of the anterior horns were diminished in size and not so numerous; but one gets the impression that the diminution in number was not very great. Baudouin says: "Mais dans les groupes externes, où elles sont plus volumineuses, elles ne dépassent pas 25 microns de diamètre et sont plus rares qu'a l'état normal."

In Lereboullet and Baudouin's case the brain and cord were normal; no change in the cells of the anterior horns nor in the anterior or posterior roots was found. This case resembles that of Spiller in its findings. Some of the muscle fibers were very small, and some were very large.

In Collier and Holmes' report (case of Collier and Wilson), as well as in that of Rothmann, the changes were pronounced in the nervous system as well as in the muscles.

Reyher and Helmholz's findings were important, showing decided changes in the muscles, but no reference is made to the nervous system.

Microscopic examination of portions of muscle excised during life are reported in the cases of Bing, Collier and Holmes (first case), Openshaw, and Skoog. In the first there were practically no changes found; in the other 3 cases they were decided. These reports are of value as far as they go, but throw no light upon the frequency of involvement of the nervous system.

A finding, so far as we know, observed only in our case (Charles K.), was the formation of fat within the muscle fibers, indicating that actual degeneration was progressing, and that the muscle change was not merely arrest of development.

From this review of the few cases as yet reported with necropsy, we may conclude that in the lighter cases (Spiller, Lereboullet and Baudouin) the alteration may be confined to the muscles, and possibly, therefore, the disease is primarily muscular, but this inference is not fully warranted. In the intense cases the nervous system is affected in marked degree. The relations to muscular dystrophy, as maintained by Batten, and to the Werdnig-Hoffmann type of muscular atrophy, as so ably set forth in Rothmann's excellent paper, are as yet undetermined. The points of resemblance to both these diseases cannot be ignored. Cassirer[19] in his recent paper has described amyotonia congenita so fully that little is left to be said upon the subject. A review of the literature is given by him, as also in the early paper by one of us (Griffith[20]), and of later cases in the present contribution.

We are indebted to Dr. Allen J. Smith for the photomicrographs.

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[19] Loc. cit.

[20] Loc. cit.

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A PATHOLOGICAL STUDY OF TÜRCK'S BUNDLE.*

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The exact anatomical relation of the fasciculus of Türck is still a matter of dispute. It has been variously described as faisceau de Türck (Meynert),[1] faisceau sensitif (Charcot,[2] Ballet[3] and Brissaud),[4] faisceau cortico-protubérantiel postérior (Flechsig),[5] faisceau occipito-temporo-protubérantiel (v. Bechterew),[6] occipito-temporale Brückenbahn (Flechsig),[7] temporale Brückenbahn (Obersteiner),[8] and parieto-temporal Brückenbahn (v. Monakow).[9]

It was thought by Meynert (in Dejerine) to be the sensory portion of the pyramids and that this bundle of fibers connected the posterior columns of the spinal cord with the occipital lobe through the medium of the nuclei of Goll and Burdach. According to Brissaud it consisted of sensory fibers which Ballet believed occupied the external part of the cerebral peduncle. Charcot believed that it was made up of centripetal fibers which were prolongations of the sensory spinal tracts and that it was never the seat of secondary degeneration. Obersteiner stated that the "temporale Brückenbahn" remained preserved in secondary descending degeneration of the peduncle though in exceptional instances of extensive descending degeneration it may be implicated, a fact which spoke against a purely centripetal origin. That the function of these tracts is sensory, Dejerine however contended was not proven clinically or experimentally.

The cortical origin of this bundle was first established by v. Bechterew (1887), and later by Dejerine[10] (1893), and Kam[11] (1895). This was later confirmed by Ferrier and Turner[12] (1898) who experimented on monkeys, and Pusateri[13] (1898) who experimented on cats. Still more recently Long[14] (1899) contended that the "faisceau de Türck" was a descending tract.

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* Read at the thirty-seventh annual meeting of the American Neurological Association, May 11, 12 and 13, 1911.

From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania.

According to Dejerine the fasciculus of Türck was related to the middle portion of the temporal lobe, especially the cortex of the second and third temporal lobes. Kam also believed that these fibers came from the temporal lobe and Brodman[15] asserted that the fasciculus of Türck was a projection bundle extending between the "basal" temporal convolution and the pons. On the contrary, Flechsig[16] was convinced from extensive studies of descending degeneration that Dejerine's opinion was incorrect.

Kattwinkle and Neumayer[17] also concluded from their study of the subject that Türck's bundle was composed of centrifugal fibers which came mainly from the third, some from the second and less from the first temporal convolution.

Van Gehuchten[18] (1906) did not believe that the existence of a descending "acoustic" pathway was definitely established, though he admitted that the fasciculus of Türck connected the auditory sphere with the nuclei of the pons.

That this bundle of fibers came from the temporal and occipital lobes was claimed by v. Bechterew (1887), Flechsig, Kreuser,[19] Meynert, Zacher[20] and Herwer.[21] Winkler,[22] Jelgersma[23] and Brero[24] believed that it originated in the parietal lobes, while in the opinion of Sioli,[25] Rossolimo,[26] Frylinck,[27] and v. Monakow, it came from the parietal and temporal lobes, v. Monakow claiming that some fibers came also from the occipital lobe.

Marie and Guillain[28] (1903) studied nineteen cases and concluded that the fasciculus of Türck is a descending tract and degenerates downwards after central lesions. The region which most frequently gives rise to this degeneration is just back of the posterior segment of the internal capsule in the white substance situated between the temporal convolution and the external wall of the occipital horn of the lateral ventricle. The fibers they believed came chiefly from the third temporal convolution.

The case of Mills and Spiller[29] showed that the first and the upper anterior part of the second temporal gyrus, including a portion of its upper middle segment may be degenerated, without at the same time the fasciculus of Türck showing degeneration.

Recently Löwenstein[30] reported a case from which he concluded that the fasciculus of Türck did not originate in the frontal half of the second temporal and the frontal two thirds of the third temporal convolution, these regions having been implicated by a tumor while the fasciculus of Türck remained normal. These facts are directly opposed to the teachings of Dejerine, and taken together with the findings in Mills and Spiller's case and my own cases herein reported, would indicate that the temporal convolutions sent few, if any, fibers to the fasciculus of Türck and what fibers that may originate in the temporal lobe come mainly from the occipital third of these convolutions, for in Lowenstein's case there was no degeneration of the fasciculus of Türck, the frontal half of the second temporal and the frontal two thirds of the third temporal convolution having been destroyed by a tumor; in Mills and Spiller's case in which the frontal half and upper middle segment of the second and the first temporal convolution were destroyed, the fasciculus of Türck was intact; and in my second case this fasciculus was preserved while at the same time there was atrophy of the middle portion of the second and third and a part of the first temporal convolution.

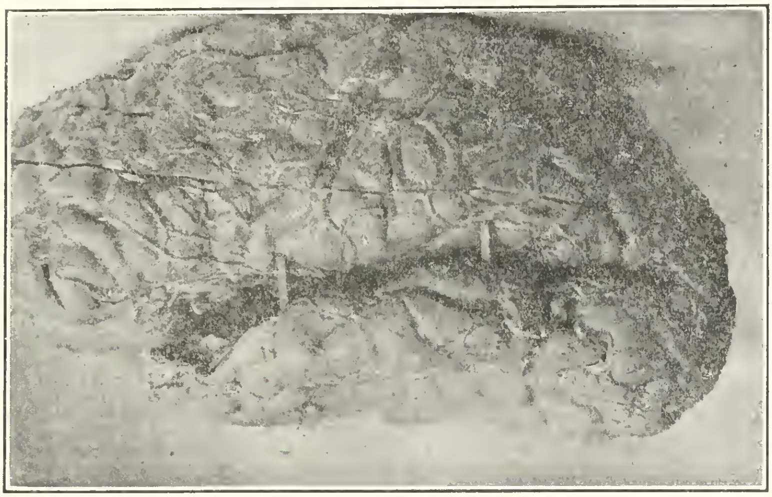

FIG. 1. Case 1. Atrophy of the temporal convolutions.

This leaves only the posterior part of the temporal gyri from which these fibers may arise.

The following two cases offered an opportunity of throwing some light on this subject, and while the findings are not unusually novel, in view of the fact that thus far there is no uniformity of opinion regarding the origin of the fasciculus of Türck, they add data of some importance in the study of this subject.

Case I.—As a result of thrombosis which obliterated almost totally the right middle cerebral artery there was found atrophy of the first and second temporal gyri. The third temporal gyrus appeared to be intact on gross inspection (Fig. 1).

There was also observed on cross section, a cystic atrophy implicating the external capsule, the internal capsule, the optic thalamus in large part and the posterior two-thirds of the lenticular nucleus (Fig. 2).

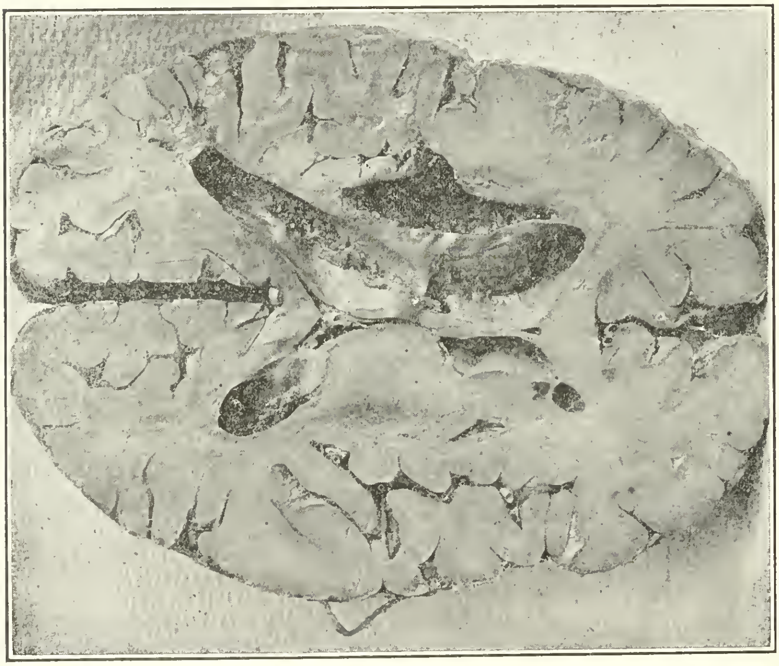

Fig. 2. Showing atrophy implicating the external capsule, internal capsule, optic thalamus, and posterior two-thirds of the lenticular nucleus.

The right half of the brain was cut in serial sections from below upwards to the upper limit of the atrophy.

Sections 34 and 70 represented the third temporal convolution and showed no atrophy, but the white matter stained very poorly. There is at the anterior external tip of the second temporal convolution which was present in these sections, a slight atrophy, while the white matter of the second temporal convolution stained poorly.

In section 108 there was slight atrophy of the posterior portion of the third frontal and the anterior portion of the second temporal convolutions.

In section 131 corresponding to Fig. 233 in Dejerine, there was atrophy of the whole temporal lobe which corresponded to the anterior portion, to the first temporal in its middle portion, to the second temporal and posteriorly to the third temporal convolution, and reached as far back as the anterior occipital fissure of Wernicke. The fusiform lobe was atrophied and the lateral ventricle was much distended.

In section 149 the level corresponded to Fig. 222 in Dejerine. The atrophy at this level implicated the posterior two-thirds of the third segment of the lenticular nucleus. The first and second temporal convolutions at this level were extensively atrophied. The tapetum was degenerated as well as the optic radiations of Gratiolet and the fasciculus longitudinalis inferior. Some few fibers were stained in the internal capsule in the anterior and posterior segments, but in a position which corresponds to the bundle of Türck the staining was poor.

At this level of section 169, the findings were practically the same as those just described, except that the atrophy of the temporal convolutions was less evident, implicating mainly that portion of the second temporal convolution which lay 4.5 cm. in front of the anterior occipital fissure of Wernicke. The first temporal convolution anterior to this was preserved in part, but the white matter did not take the stain. The lenticular nucleus was destroyed totally in its posterior two thirds and the external capsule and the claustrum were almost completely atrophied, as also was the white matter between the pulvinar and the retrolenticular segment of the internal capsule, and the internal and external medullary layers of the lenticular nucleus.

It will therefore be seen that the atrophy thus far implicated the first and second convolutions mainly, especially in their posterior segments.

In section 189, practically the same conditions existed. At this level where the fasciculus of Türck should be seen, there could be observed a few stained fibers only.

In section 204 the internal capsule did not take the stain at all and here the atrophy of the temporal convolutions was confined to the posterior portion of the second temporal convolution extending over an area 2.5 cm. in length. The second area of atrophy implicated the entire lenticular nucleus and extended on one side to the outer limit of the posterior limb of the internal capsule which was completely degenerated, and involved in part the anterior limb of the internal capsule. The outer limit of atrophy extended almost to the gray matter of the temporal lobe.

In section 209 the same conditions prevailed except that the atrophy of the temporal lobes was less apparent. There was intense degeneration of the optic thalamus, but the central area of atrophy was not so marked.

In section 238 the second parietal convolutions were present; but the white matter of these stained poorly.

The sections higher up showed nothing pronounced or definite except that the white matter of the third frontal and parietal convolutions stained poorly.

It is difficult to see in a study of these specimens how any fibers from the third temporal convolution could reach the level of the bundle of Türck on account of the extreme atrophy in the region of the lenticular nucleus and because the white matter of the third temporal gyrus was in great part degenerated. The white matter of the third temporal convolution was so degenerated that practically no healthy fibers could be demonstrated. Some few fibers might have reached that level from the basilar surface but it would be misleading to draw any conclusions from this case unless we were to include all three of the temporal convolutions as being cut off from the retro-lenticular segment of the internal capsule which is the situation of the fasciculus of Türck, as the few fibers which may have reached to this level from the third temporal convolution could not have been in sufficient numbers to be demonstrable.

In the pons at the level of the red nucleus, the foot of the peduncle on the left side was almost completely atrophied. A few fibers took the stain at the extreme internal end of the foot. At the external portion of the foot of the peduncle corresponding to the position of the fasciculus of Türck, a very few fibers took the stain but were mainly if not entirely made up of transverse fibers (Fig. 4, *A*).

The foot of the peduncle on the opposite side stained very well. The anterior pyramid on the left side in the medulla oblongata was completely degenerated, and the crossed pyramidal tract in the spinal cord on the right side was degenerated.

Summary.—In this case the three temporal convolutions were practically entirely atrophied or degenerated and the atrophy cut in large part the optic radiation of Gratiolet and the fasciculus longitudinalis inferior. The fasciculus of Türck was totally degenerated in the foot of the peduncle though there were a few stained fibers demonstrable in the subthalamic region and some fibers which were probably not longitudinal ones in the foot of the peduncle.

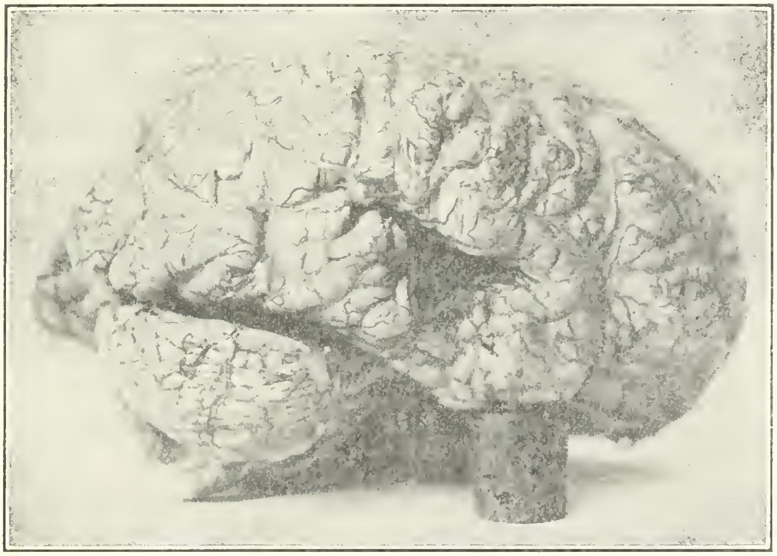

Fig. 3. Case 2. Atrophy of the temporal convolutions in the middle portion.

Case II.—There was atrophy of the middle portion of the second and third temporal convolutions (Fig. 3). The anterior limit of the atrophy was 2.5 cm. posterior to the anterior border of the temporal gyri. In its upper portion the atrophy measured antero-posteriorly 4.5 cm. and implicated partially the first temporal gyrus in its posterior portion. The middle or second temporal gyrus was atrophied to the extent of 2.5 cm., the third temporal 2 cm., and on the basal surface this atrophy extended for about 2 cm. Serial sections were made beginning at the base of the brain and extending to the upper limit of atrophy.

In sections 20 and 50 there was observed atrophy of the third temporal convolution beginning 2.8 cm. behind the anterior limit of the temporal lobe and extending for 1.2 cm. posteriorly on the outer aspect. The white matter of the anterior portion of the third temporal lobe stained poorly.

In section 74, representing Fig. 234 in Dejerine, the second temporal convolution posterior to the "sillon paralléle" was atrophied, the atrophy implicating the gray and white matter. The atrophy extended posteriorly for 3.5 cm. Anterior to this and posterior as far as the third temporal convolution, the white matter showed degeneration. At this level the descending horn of the lateral ventricle was found distended.

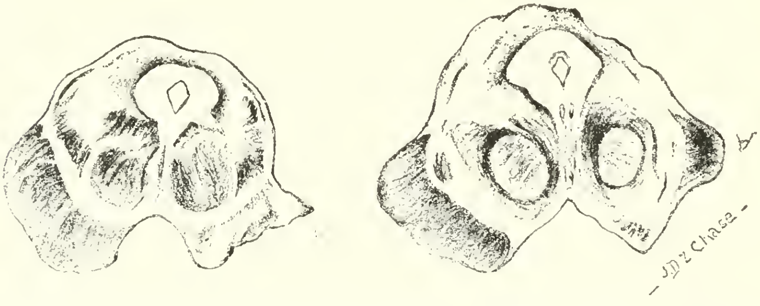

Fig. 4. Case 1. Degenerated fasciculus of Türck. Case 2. Preserved fasciculus of Türck.

In section 95, the optic radiations of Gratiolet and the fasciculus longitudinalis inferior were degenerated to a moderate degree in the anterior portion, though the latter was preserved posteriorly.

The atrophy of the second temporal convolution at this level measured 2.5 cm. posterior to the tip of the first temporal convolution, the white matter of which did not take the stain at all.

In section 110 the atrophy extended posteriorly for 3 cm.

In section 134 corresponding to level 230 in Dejerine, atrophy of the second temporal convolution was observed, the anterior limit of which is .5 cm. in front to the anterior wall of the descending horn of the left lateral ventricle. It extended posteriorly 2.5 cm. and implicated all the white and gray matter between the cortex and the wall of the ventricle. At this level which shows the third segment of the lenticular nucleus, the fasciculus of Türck made its appearance. It was not degenerated to any appreciable extent. The claustrum apparently was degenerated as well as the external capsule and the red nucleus did not stain deeply.

In section 165 the bundle of Türck was not degenerated.

In section 176 corresponding to Fig. 223 in Dejerine, it was evident that the superior border of the atrophy had been reached. Here however the white matter of the first temporal convolution was markedly degenerated.

The foot of the peduncle on the left side stained very well. On the right side, however, only a small number of fibers stained in the internal fifth and in the external fifth, while the volume of the foot of the peduncle was distinctly reduced and did not take the stain at all. The fasciculus of Türck stained well and was only slightly if at all reduced in volume (Fig. 4).

Summary.—This case showed atrophy of the middle portions of the second and third temporal convolutions with degeneration of the white matter of the first temporal convolution and slight atrophy of the posterior portion of the first temporal convolution. The fasciculus longitudinalis inferior and the optic radiations of Gratiolet were markedly implicated at the level of the subthalamic region.

The fasciculus of Türck was in main preserved, though its volume in the foot of the peduncle was perhaps somewhat reduced.

The first case shows that destruction of the white matter in the posterior portion of the posterior segment of the internal capsule, as well as that between the posterior portion of the temporal lobe and the wall of the descending horn of the lateral ventricle, is associated with complete or almost complete degeneration of the fasciculus of Türck in the foot of the peduncle. This is the region of the brain that Marie and Guillain have already described as being the favorite locality for degeneration, in association with degeneration of the fasciculus of Türck. There was not only a cutting off of the fibers from the three temporal convolutions but also of those from the occipital lobe in part.

In the second case the atrophy of the middle portion of the second and third temporal convolutions and the posterior part of the first temporal convolution, associated with degeneration of the fasciculus longitudinalis inferior and the optic radiations of Gratiolet, did not cause much if any degeneration of the fasciculus of Türck.

No very radical conclusions are justifiable from these cases, though it would seem probable that fibers from the occipital lobe did not figure greatly in the formation of the fasciculus of Türck, nor did the fibers from the middle portion of the second and third temporal or the posterior part of the first temporal convolutions.

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DISPLACEMENT OF THE CEREBELLUM FROM TUMOUR OF THE POSTERIOR CRANIAL FOSSA.[1]

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I DESIRE to call attention in this paper to two forms of displacement of the cerebellum as a result of tumour of the posterior cranial fossa—viz., the lateral displacement, in which the cerebellum is nearly at a right angle with the brain-stem; and the upward displacement, in which the tentorium is much distended and the occipital lobes are widely separated by the dislocated cerebellum.

Displacement of the cerebellum from tumour of the cerebello-pontile angle so that the cerebellum is twisted greatly toward one side is of unusual occurrence. In my collection of nine large tumours external to the cerebellum, in or near the angle mentioned, extreme lateral displacement resulted only twice. I do not include in this number tumours seen at operation, or intracranial tumours that developed in the region of the trigeminal nerve, or tumours in the cerebellum. Usually atrophy of the parts about a tumour is produced when the pressure is intense, and twisting of the cerebellum is therefore less likely to occur.

The twisting of the cerebellum complicates operation for removal of a tumour of the posterior cranial fossa. The medulla oblongata becomes less resistant, and a fatal termination is more likely to follow operation than in cases in which less distortion of the medulla oblongata has occurred. The cerebrospinal fluid also may be increased, as in my Case No. 558, reported in this paper, and the escape of so large an amount of fluid produces very grave symptoms. The twisting of the cerebellum does not necessarily hasten death, although the effect on the vagus nerve from displacement of the medulla oblongata might be regarded as serious.

In Case 1 [No. 421] the symptoms of tumour first appeared about three and a half years before death. They became intense, and although the displacement of the cerebellum was great, the lateral twisting on the brain-stem was not so extreme as in Case 2 [No. 558], in which the symptoms were much less severe. In Case 1 the tumour was much larger than in Case 2, and it must have stretched the tentorium greatly, as it extended far enough forward to press upon the left oculomotor nerve. It also depressed the left occipital lobe. A deep depression of this part of the brain is a somewhat rare occurrence from a subtentorial tumour. The displaced cerebellum separated the occipital lobes from one another, and the space provided in this way diminished the degree of lateral twisting of the cerebellum upon its axis. It is possible that during life the separation of the occipital lobes was not so great as is represented in fig. 1, as the tentorium, though greatly distended, must have afforded considerable resistance to pressure. A brief reference to this case with the photograph has been published in a previous paper by me in the *Journal of the American Medical Association*, vol. lii, No. 4, p. 278, 1909.

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[1] Read before the Philadelphia Neurological Society, January 27, 1911.

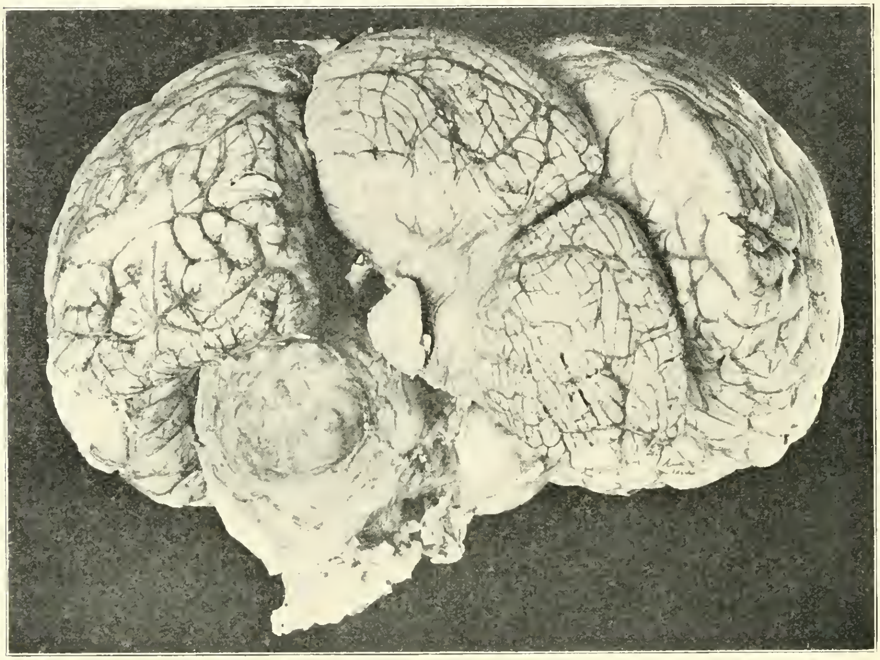

FIG. 1.
Case 1 [No. 121].—The occipital lobes are widely separated by the displaced cerebellum.

Two parts of the brain are especially affected by the distortion of the cerebellum—viz., the pons at its union with the cerebral peduncles, and the medulla oblongata. If the pressure be from below upward with sufficient force to separate the occipital lobes and stretch the tentorium, as in Case 1, the lateral displacement of the cerebellum is less and the stretching of the medulla oblongata also is less. When the pressure is from one side and displaces the cerebellar lobes almost to a right angle with the brain-stem, as in Case 2, the stretching of the medulla oblongata is pronounced, and this part of the brain may be displaced as

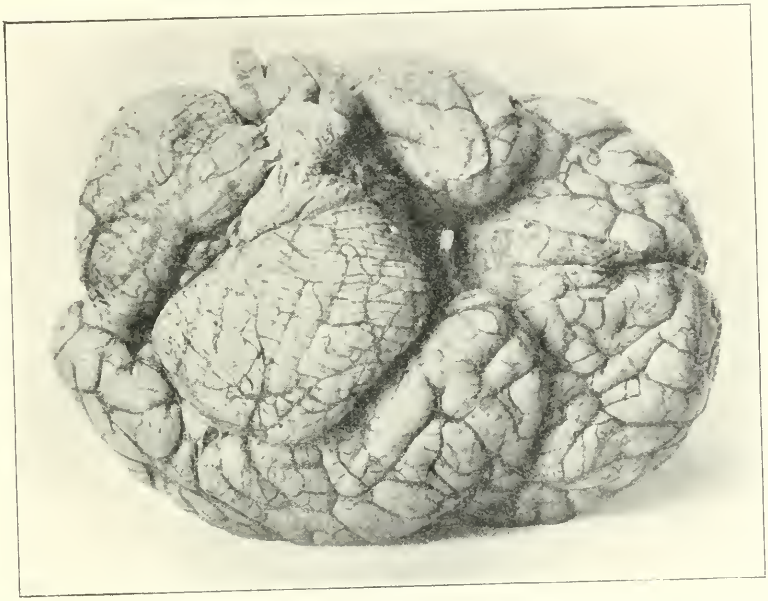

Fig. 2.

Case 2 [No. 558].—The tumour is on the right side, and has pushed the cerebellum to the left. The pons and medulla oblongata are displaced 3·5 cm. to the right (the side of the tumour) by the left lobe of the cerebellum.

much as 3·5 cm. Inasmuch as the medulla oblongata is continuous with the cervical cord, a dislocation of such a degree would cause stretching of the nerves arising from the former. In Case 2 the cerebellum was pushed to the left by the tumour, but the pons and the medulla oblongata were pushed by the displaced left cerebellar lobe to the right—i.e., to the side of the tumour—and the left side of the pons was much flattened by the pressure from the left cerebellar lobe.

In some instances symptoms have been observed indicative of a lesion on the side opposite to that occupied by a basal tumour. An explanation has been sought in pressure upon the medulla oblongata on the side of the tumour, below the motor decussation, or in pressure of the pons and medulla oblongata of the opposite side against the base of the skull, or, more recently by Oppenheim, in the constriction of the medulla oblongata by the tightly stretched vertebral artery. Oppenheim has considered it possible that in lateral displacement of the pons and medulla oblongata, the pyramidal tract on the convex side of the arch—i.e., the opposite side to the tumour—may be more damaged. I am not aware that anyone has referred to stretching of cranial nerves on the side opposed to the tumour by the displacement of the pons and medulla oblongata from the median line in the direction of the tumour by a dislocated cerebellar lobe, or to pressure on the contralateral side of the pons by the displaced cerebellar lobe, as seen in Case 2.

The pain in Case 2 was described as unbearable, as of a pressing or burning character, and as though someone were pressing on the side of the head. It was situated in about the left motor region, but extended well into the left occipital region. Very little pain was felt in the left side of the face. The sensation was more one of burning than of pain. The supra-orbital and infra-orbital foramina were tender to pressure on both sides. In speaking, and voluntary drawing up the corners of the mouth, together or separately, the left side of the face was not moved so much as the right, but the inequality of the movement was slight. Hearing for the voice and watch was considerably impaired in the left ear, though much less than in the right ear.

It is possible that the intense pain in the left side of the head, the slight weakness of the muscles innervated by the left facial nerve, and the moderate degree of deafness in the left ear, were caused by stretching of the left trigeminal, facial, and acoustic nerves from the displacement of the medulla oblongata and pons 3·5 cm. to the right—i.e., toward the side of the tumour. This displacement was produced by pressure from the distorted left lateral lobe of the cerebellum, and is very different to the displacement referred to by Oppenheim, which is to the side opposite to the tumour.

It is uncertain whether headache can be caused by stretching of the trigeminal nerve. It seems at least possible, as the nerves of the cerebral dura, according to "Gray's Anatomy," are filaments from the trochlear, the ophthalmic division of the trigeminal, the semilunar, or Gasserian ganglion, the vagal, the hypoglossal, and the sympathetic. In Case 2, the supra-orbital and infra-orbital foramina on each side were tender to pressure. The stretching of the left trigeminal nerve may have caused this tenderness on the left side, and the implication of the right trigeminal nerve in the tumour at its surface doubtless caused the tenderness on the right side.

One is impelled to ask why it is that lateral displacement of the cerebellum does not occur more frequently.

In a case of Dr. Mills' [No. 342] the tumour was very much larger than in Case 2, and was in the same situation, but it formed dense adhesions with the left cerebellar lobe and pons, and caused much atrophy of these parts. It did not cause any important displacement of the cerebellum. It was as firm a tumour as that in Case 2, and grew in the same direction, along the margin of the cerebellar lobe. It caused moderate atrophy of the overlying occipital lobe. The adhesions of the tumour to the surrounding parts were much stronger than were those of Case 2, and probably in this fact lies the explanation of the absence of displacement of the cerebellum. The pressure on the cerebellum from so large a tumour (measuring 5·5 cm. antero-posteriorly by 3·5 cm. laterally) must have been greater than from the tumour in Case 2, therefore neither position nor size of the growth is sufficient alone to explain the greater displacement of

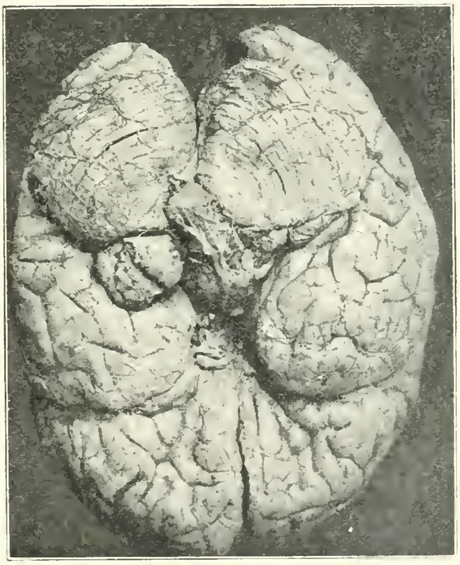

Fig. 3.

Case 3 [No. 205].—A small tumour having a similar origin to that in Case 2, but causing no displacement of the cerebellum.

the cerebellum in Case 2, but between these two tumours a difference exists in the density of the adhesions between the tumours and the surrounding parts. If adhesions form early between the tumour and the cerebellum on one side, and the tentorium on the other, the likelihood of much displacement of the cerebellum is lessened, and atrophy of the parts about the tumour will occur. The cerebellum being bound to the tumour by dense adhesions resists displacement, and atrophies proportionately to the pressure from the tumour. Other factors are concerned, however, in the displacement of the cerebellum.

In Case 3 [No. 205], one of Dr. Mills', with whom I saw the patient (reference has also been made by Dr. Weisenburg to this case), the tumour was about the size of that in Case 2, was equally firm and equally well defined from the surrounding tissues. It occupied a very similar position, but, unlike the tumour of Case 2, it caused considerable atrophy of the overlying occipito-temporal lobe, this through the tentorium, and only moderate atrophy of the adjoining cerebellar lobe. This tumour measured 2·5 cm. antero-posteriorly by 3 cm. laterally, but where it arose from the cerebello-pontile angle it measured only 1·5 cm. The greater diameter was at its outer portion, where it sent a projection anteriorly which caused pronounced atrophy of the occipito-temporal lobe. The shape of the tumour as well as the atrophy of the occipito-temporal lobe, which could only have been produced by distension of the tentorium, indicates that the pressure from the tumour must have been chiefly forward.

The resistance afforded by the tentorium must be another factor in the displacement of the cerebellum; where the membrane is dense and unyielding, the pressure against the cerebellum is greater.

In Case 4 [No. 338], also one of Dr. Mills', the adhesions between the cerebellum and the tumour were very dense, and great atrophy of the cerebellar lobe on the same side and of this side of the pons occurred. The cerebellum had not been displaced laterally, although this tumour measured 5 cm. antero-posteriorly by 6 cm. laterally by 4 cm. from above downward. The pressure evidently was from below upward, for while only slight atrophy of the overlying occipital lobe had been caused, this occipital lobe had made a deep indentation in the upper part of the lateral lobe of the cerebellum on the side of the tumour by the pressure of the inferior and median edge of the occipital lobe, and the cerebellum had been pushed upward between the two occipital lobes, separating the occipital points as much as 6 cm. The pressure from the displaced cerebellum on the median side of each occipital lobe must have been considerable, not so much, however, as in Case 1, where the occipital points were separated as much as 8 cm.

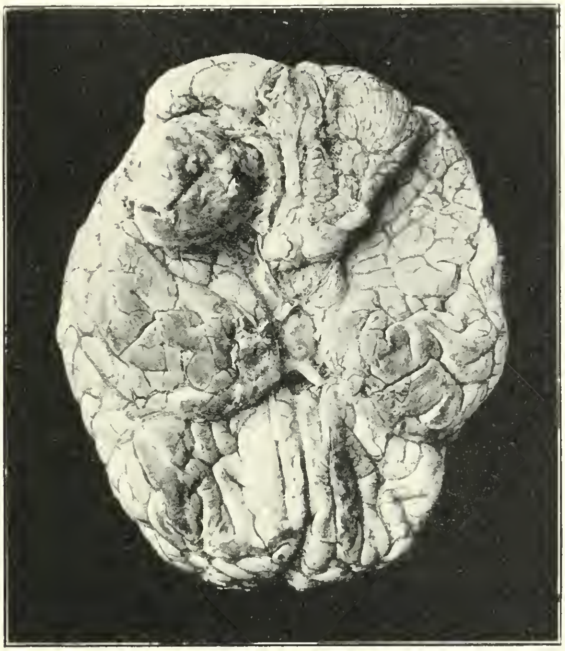

Fig. 4.

Case 4 [No. 338].—A much larger tumour than that in Case 2, and having a similar origin, but causing little displacement of the cerebellum.

In Case 5 [No. 296] where the patient was under my care the separation was even greater. A large tumour measuring 5 cm. antero-posteriorly by 6 cm. laterally by 5 cm. from above downward, developed in the cerebello-pontile angle, but its origin doubtless was from the dura at the base of the posterior cranial fossa. It was not adherent to the brain. The pressure being from below upward, it caused no lateral displacement of the cerebellum, but it drove the cerebellum far upward between the occipital lobes, separating the occipital points 9 cm. Such displacement as this, notwithstanding the intervening tentorium, gives a fair idea of the great pressure that must have existed, and suggests the possibility of occipital lobe symptoms from cerebellar tumour. As the displacement, however, cannot occur until the pressure from the tumour becomes great, and not until papillœdema has developed, hemianopsia could not be expected, and symptoms of occipital lobe lesion would be masked.

Condensed notes of the two cases selected as illustrative of the two forms of displacement described in this paper are as follows :—

Case 1 (laboratory number 421).—E. M., female, aged 20, was admitted to the Philadelphia General Hospital, May 1, 1907, and died January 8, 1908. Her sight had been poor since she was 7 years old. Frontal headache had existed about three years, but it had become much worse during the two months before her entrance in the hospital. Two weeks before any notes were taken she began to have difficulty in walking and in speaking. When she came to the hospital she was unable to walk or stand alone, and had a tendency to fall backward, but the voluntary power in the lower limbs was fair. She was very ataxic even in the heel-to-knee test. The patellar reflexes were exaggerated, and ankle-clonus and the upward movement of the toes in the Babinski test were obtained on each side. The movements of the upper limbs were ataxic, and the grip of each hand was weak. The biceps reflex was exaggerated on each side. The voice was whining. Weakness of the lower part of the right side of the face was present. The tongue deviated to the right when protruded. The right masseter muscle was weak, and the lower jaw deviated slightly to the right when the mouth was opened. These symptoms evidently were caused by pressure upon the left cerebral peduncle and left upper part of the pons. Reaction of the iris to light and in convergence was lost in each eye, and the left eye protruded. Swallowing was difficult. Mentality became much impaired, and stupor pronounced.

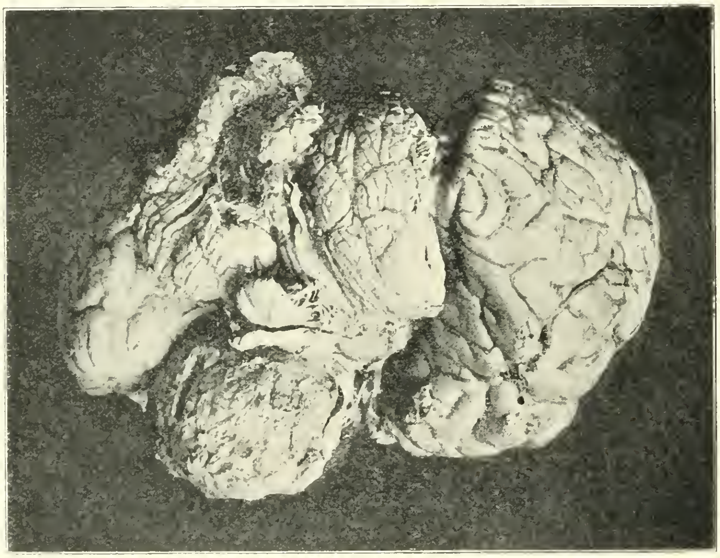

Fig. 5.

Case 5 [No. 296].—A tumour of the posterior cranial fossa. It has pushed the cerebellum between the occipital lobes as did the tumour in Case 1.

The body was in the cerebellar attitude described by Hughlings Jackson. Dr. Hansell found the late stage of high-grade optic neuritis passing into atrophy in both eyes. The left eyeball could be moved only slightly, and the movement preserved was downward, and slight ptosis was observed on the left side. Late in the disease the right eyeball deviated outward. The right upper limb was a little weaker than the left. Hunger was pronounced, and a second dinner was demanded nearly every day. The upper limbs became very rigid, the fingers were flexed into the palms and the forearms flexed on the arms across the chest. The lower limbs also became very rigid in extension and the feet over-extended.

A tumour measuring 9 cm. antero-posteriorly by 6 cm. laterally was found in the left cerebello-pontile angle. It was very nodular and well defined. It evidently had arisen from the dura at the base of the brain, and had affected the nerves coming from the left side of the medulla oblongata and pons by pressure only. The cerebellum had been greatly displaced laterally, but not so much as in Case 2, the edge of the right lateral lobe of the cerebellum not so nearly forming a right angle with the brain-stem. The tumour had forced apart the occipital lobes, and had depressed the basal part of the occipito-temporal lobe. It must have stretched the tentorium greatly. The greater part of the periphery of the tumour, especially the upper part, was covered by a dense fibrous membrane, and a piece of tentorium extended medianly from this membrane. The tumour pressed severely upon the left third nerve, but the right third nerve was not implicated. The posterior and anterior horns of the left lateral ventricle were much compressed. The right lateral ventricle was dilated at all parts. The tumour was a fibro-sarcoma.

Case 2 [laboratory number 558].—S. W., female, aged 33, was seen by me in consultation with Dr. H. D. Beyea, December 22, 1910. She had had severe headache for thirteen months. She had nausea and vomiting occasionally, and her condition had become much worse following an operation for retroflexion of the uterus three weeks previously. The pain in the head was almost unbearable and was of a burning character, and the head felt as though there were internal pressure. This pain was distinctly more intense on the left side, although it extended at times to the right side. Tinnitus like escaping air was heard in the right ear, and hearing was much impaired in the right ear; it appeared to be impaired also in the left ear. Pain had been felt frequently on the right side of the face. She had vertigo at times.

The patellar reflexes were exaggerated. She was ataxic on standing and walking, and the ataxia was increased by closing the eyes. Slight nystagmus was present in looking to the right.

Dr. B. A. Randall reported, December 23: "There is defect of hearing on the right side of moderate degree only for all tones, more marked for the weaker high or low tones. This corresponds with a lesion of the acoustic nerve, but in contradiction she claims transmission through the bone to the affected right ear."

Dr. Langdon, in Dr. de Schweinitz's clinic, on December 23, 1910, found

3 dioptres of swelling of the optic disc in each eye, and numerous hæmorrhages on and near the discs. On December 27 he found that the papillœdema had increased until the apex of each disc was 5½ dioptres. In addition, several fresh hæmorrhages had formed.

The reflex of the right scleral conjunctiva and, to a less extent, of the right cornea was diminished. Diadokokinesis was good in each hand. Finger-to-nose test showed slight ataxia only on the right side. Asynergia was moderate; when the patient was told to bend the head and body backward she at first was not inclined to flex the knees, and nearly fell backward. When told to lift the upper part of the body, with the upper limbs folded across the chest, she was unable to do so at first, as she raised the lower limbs from the bed instead of pressing them against the mattress.

The most serious symptoms, therefore, were nausea, vertigo. headache (more on the left side), pain at times in the right side of the face, which was with difficulty to be distinguished from the headache, as it was not present at the time of examination: diminution of the right conjunctival and corneal reflexes; tinnitus and deafness, probably of nerve origin, in the right ear; rapidly developing papillœdema with numerous hæmorrhages; ataxia on standing and walking; exaggeration of the tendon reflexes of the lower limbs, that varied in intensity from one examination to another: ataxia in the finger-to-nose test and a moderate amount of asynergia.

My diagnosis was of tumour in the right cerebello-pontile angle, but as the deafness of the right ear was not positively of nerve origin, a tumour of the cerebellum could not be excluded. The right facial nerve showed no defect.

An opening of large size was made over the occipital region by Dr. C. H. Frazier, and the dura was slit. Much cerebrospinal fluid escaped. It was thought advisable to perform the operation in two stages, but the patient did not rally sufficiently to justify any further surgical procedure, and died within a few days.

A tumour, measuring 4 cm. antero-posteriorly by 2·5 cm. laterally, was found in the right cerebello-pontile angle. It was well defined, nodular, and at no part infiltrated the brain. It had displaced the cerebellum almost to a right angle with the brain-stem. The left lateral lobe of the cerebellum indented the pons. The right trigeminal nerve was attached to the tumour at its periphery, but was not involved within the tumour. A bundle of nerve-fibres, having the origin of the acoustic nerve, entered the tumour and was lost within it. It was impossible to determine without dissection whether this was the acoustic or facial nerve, or both, but no trace of any other nerve that could be either of these was found. The tumour pressed upon the right side of the medulla oblongata and flattened this side, and may have stretched the glosso-pharyngeal and vagal nerves. The pressure of the tumour upon the pons and medulla oblongata had not caused any distension of the lateral or of the third ventricle. The tumour was of the same character as that of Case 1, viz., a fibro-sarcoma. Mallory, however, regards these tumours as endotheliomata.

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CHRONIC HYPERTROPHIC SPINAL PACHYMENINGITIS[1]

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The immediate stimulus to the writing of the present article was our experience with a case of unusual interest, admitted to the hospital of the University of Pennsylvania, May 28, 1910. This case, before it came under the observation of the writers, had presented active symptoms for about one year, during which time several diagnoses had been made. The one most favored for several months by the physicians in attendance was that of grave hysteria, this diagnostic view having been reached largely because of the great variability in the symptoms presented, and also because of some mental peculiarities, which had developed during the progress of the case, these being chiefly a tendency to unreliability of statement and great suggestibility in connection both with the relief and the production of symptoms. The experience on which this paper is based, however, is by no means exclusively confined to this instructive and unusual case.

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[1] Read at the thirty-seventh annual meeting of the American Neurological Association, May 11, 12 and 13, 1911.

Early in the professional experience of one of us a case of pachymeningitis, localized to the lower bulbar and upper cervical region, was diagnosticated during life as one of hydrophobia, necropsy showing its real nature. The bulbar forms or bulbar extension of hypertrophic spinal pachymeningitis are now comparatively well known, although the number placed on record is limited.

In another case recorded clinically during the life of the patient, although under an incorrect diagnosis, hypertrophic spinal pachymeningitis, confined chiefly to the lower cervical and thoracic region was discovered at necropsy.

An interesting case reported by Dejerine and Tinel shows the importance of an early diagnosis of pachymeningitis as well as its difficulty. In this case the meningitis had extended until it involved pontile, bulbar, and cerebellar parts. The Brown-Séquard syndrome was present with cerebellar symptoms, with epileptiform seizures, vomiting, and papillary stasis, resembling brain tumor more than a pachymeningitis. The absence of cranial nerve involvement with such extensive basilar thickening was remarkable in this case.

Dr. Mills and Dr. Spiller placed on record a case of generally diffused external pachymeningitis, but this represented a different type from that recorded in the present communication.

At the last meeting of this association in a paper on tumors and cysts of the spinal cord, Dr. Mills recorded a case of diffuse spinal meningitis with a subpial cyst, and referred also to other cases of this description which had been reported abroad and in this country, notably by Schlesinger, Horsley, Bliss, Krause, Oppenheim, Spiller, Musser and Martin; and Weisenburg. The cases referred to by these writers, however, were different in character from the subject of the present communication. They represented in part or together, cases of the so-called circumscribed serous meningitis. The point to which we wish to direct attention, however, is that in most of these cases the meningitis present was widely diffuse and was in some instances, at least in part, of the form of an internal hypertrophic pachymeningitis, adhesions having formed in single and separated portions of the inflamed membrane.

The present case had in its symptomatology much the appearance of the classical cervical hypertrophic pachymeningitis of

Charcot and Joffroy. Certain features in its symptomatology, however, which became more evident after operation and necropsy, served to separate it from the original Charcot-Joffroy type, and the presence of these features is among the reasons which would seem to call for the careful recording of the case. Atrophy and loss of power in the trunkal muscles were, for instance, among the symptoms.

Among the features of the case which call for special review are those concerned with the explanation of the variability in the symptoms, and the peculiarities in the sensory disturbance. The resemblance of the case to a cervico-thoracic tumor, to a somewhat aberrant form of syringomyelia, and to amyotrophic lateral sclerosis, is a matter of some interest. The existence of rudimentary cervical ribs is also of passing interest.

No feature in this case is of greater clinical interest than the variability of the symptoms presented by the patient up to two or three weeks before operation. This variability in symptomatology led to at least two erroneous diagnoses—at an early date that of hysteria, and later that of circumscribed serous meningitis. Almost a year before her death the patient was for a time bedridden, apparently unable to stand or walk, but chiefly under the influence of strong suggestion regained her feet and continued to stand and walk with occasional short periods of relapse, until six weeks before her death. The deep reflexes in the lower extremities presented so extreme a variation that on one day foot clonus and the Babinski reflex were present and persistent, while on the next or within a day or two these phenomena could not be elicited. Even the distal portion of the upper extremities varied considerably from day to day. The sensory symptoms also showed some variations and differences, both in the upper extremities and in the trunk and lower limbs.

The best explanation of these variations seems to be indicated by the microscopical findings.

The widespread obliteration or partial obliteration of the vessels of the membranes and cord would have caused much variation in the blood supply under the influence of general vascular states; even those vessels which were patulous were thickened and inelastic and would have failed to respond in a compensating way for differences in general blood pressure. The number of spinal cells having been greatly reduced, local areas in the cord would also have been much more readily fatigued.

Cases of chronic hypertrophic pachymeningitis may, as in this instance, lead to difficulties in their diagnosis, the diseases most resembling it being amyotrophic lateral sclerosis, syringomyelia, multiple neuritis, cord tumor, circumscribed meningeal cyst, and supernumerary ribs. Hysteria, while diagnosticated as such in our own case, should not cause any difficulty where such distinct organic signs occur either early or late as were present. Hysterical symptoms might occur concomitantly, but should not obscure the organic condition present.

Amyotrophic lateral sclerosis presents a picture resembling in some respects that of pachymeningitis, but the absence of pain in amyotrophic lateral sclerosis is a distinguishing sign, and when pain does occur it is not intense and is due to fatigue or overstretching of muscles and joints, and cannot be definitely ascribed to nerve distributions. Paresthesias and objective changes in the senses of pain, temperature, and touch, are not found in this disease, although of frequent and often of early occurrence in pachymeningitis. Bladder disturbances if present are entirely against any diagnosis of amyotrophic lateral sclerosis.

Symptoms common to syringomyelia and chronic hypertrophic pachymeningitis are muscular atrophy and loss of power with loss of reflexes in the motor arcs affected, deep reflexes increased in those below the affected arcs, and objective sensory changes. These sensory changes may be to some degree dissociated in pachymeningitis, but generally they are not so marked or so distinctly defined as in syringomyelia. In the former, also, a preponderance of subjective sensory symptoms will be found as a rule, such as intense pain and paresthesias along the distributions of the nerves involved. A certain degree of spinal rigidity occurs in both these affections, but it is more marked in pachymeningitis. The atrophy in both cases involves more often and more extensively the muscles of the hands and forearms, with greater tendency to further extension in pachymeningitis and with greater trophic changes in syringomyelia. The so-called succulent hand (main succulente) of the French writers does not occur, or at least very rarely in pachymeningeal cases.

Multiple neuritis might be confused with a case of pachymeningitis at its incipiency, as pain is then a most prominent symptom in both diseases. The excessive tenderness of the nerve trunks in neuritis with the absence of early objective sensory signs would aid in the differentiation, while the later occurrence of muscular atrophy would add to the certainty of diagnosis of pachymeningitis. In multiple neuritis a decrease of the deep reflexes of the lower limbs occurs in contrast to the usual increase in pachymeningitis. A history of some predisposing or exciting factor in neuritis is of importance, such as previous alcoholism, infectious fevers, metallic poisons, etc. A history of lues or traumatism is of some etiological significance in cases of pachymeningitis.

Tumors of the cervical cord in some instances give symptom complexes very difficult or even impossible to distinguish from that of pachymeningitis. Muscular atrophies and loss of power and reflexes with increased reflexes below the affected arcs, paresthesias and anesthesias with subjective pain, all are prominent symptoms in both diseases. The pain is possibly more excruciating and sharper or more lancinating in tumor cases, while those of pachymeningitis often have some rigidity and soreness of the neck and aching of the shoulders preceding the violent pain by a period of months or even years. The occurrence of segmental distribution of objective sensory changes points more to tumor. The X-ray is of value in showing a new growth, though not always certain in the result.

In circumscribed cysts the picture may resemble that of tumor, and differential points of the former would be of almost equal value for this condition. However, pain here is usually of less intensity and often somewhat fluctuating. The fluctuation of the symptoms has been supposed by some to be pathognomonic of circumscribed cyst, but this rapidity in the change of symptoms occurred in our case. Any case of pachymeningitis could readily form a cyst, thus causing a compound condition. Motor loss and atrophy as a rule are not so marked in cases of circumscribed cyst as in those of tumor or pachymeningitis.

In cases of supernumerary ribs any muscular atrophy or other trophic changes, loss of power, or sensory changes are due to peripheral nerve or plexus involvement. No increase of deep reflexes in the lower limbs occurs, and no sign of organic cord or meningeal lesion such as pains and sensory changes in trunk or legs, and the presence of ankle clonus and dorsal extension of the great toe upon irritation of the sole of the foot. Interference with vesical and rectal control also never occurs in cases of cervical ribs. The presence of the bony overgrowth may often be easily palpated, and is always distinctly seen in radiographs. Weakness in one or both radial pulses is very common, and this weakness becomes more distinct upon pulling back the shoulders and arms so as to cause pressure of the cervical ribs upon the subclavian arteries.

Cervical tabes, a condition of rare occurrence, although frequent enough to enter into consideration, has a complex of symptoms greatly resembling the present case. The shooting pains in the arms with the lost reflexes and presence of paresthesias or even areas of objective sensory changes, might point toward either condition. Further, a difference in the pupillary reactions of the two sides might occur in pachymeningitis where the sympathetic fibers are involved at their exit from the cord, but a typical Argyll-Robertson pupil would not be obtainable and there might be other evidences of unilateral sympathetic involvement. The absence of knee-jerks and Achilles reflexes would be a feature of tabes strongly opposed to the heightened lower limb reflexes of pachymeningitis cervicalis hypertrophica, although the loss of reflexes is often lacking in cases of tabes cervicalis. Ataxic conditions of the arms would probably be more marked in tabes. Romberg's sign with areas of hypesthesia, bladder and rectal disturbances showing involvement by tabes of the lower parts of the cord, could later occur and clear the diagnosis, while ocular palsies and optic nerve atrophies are of further diagnostic significance.

The question of operative treatment in cases of this sort is of much practical importance. While no harm was done and death was here probably deferred rather than hastened, nevertheless the results to be obtained from surgical procedure in such a case are not sufficient to warrant it. The operation in this case was decided upon chiefly with the hope of discovering a circumscribed serous meningitis or a cervical tumor or pachymeningitis so localized as to be removable.

The case summarized is as follows: The patient was a woman about forty-five years old. For several years she had had occasional pains at the lower part of the back of the neck, and between her shoulders, and thirteen or fourteen months before she came under observation she first experienced severe pain in her back between the shoulders, followed in a few days by weakness in the right arm and hand. About two weeks later she lost power in the lower limbs so that she was unable to walk, the right limb being more affected than the left. After a few weeks she recovered power in the lower limbs, losing it again a few weeks later. Seven months later she had an attack of severe pain in her head followed by nausea and vomiting and a convulsion. Her position in this and in some subsequent convulsive attacks, was that of opisthotonos. She would remain unconscious or partially so for several hours. Altogether she had a half dozen of such attacks. During five months before she was first seen she showed a curious condition of varying power in the lower extremities, being sometimes unable to walk, while at others she could go about a room or the house holding on to furniture or other supporting objects. This variability in her power to use her lower extremities was so marked that her attending physician was strongly inclined to believe that the case was one of hysteria.

Examination showed mentality clear but memory defective. The patient was inaccurate in her statements.

The ocular movements were free in all directions. The pupils were slightly dilated, the left being a little larger than the right. Both reacted promptly to light and accommodation.

Examination showed no impairment of the muscles supplied by the 5th or 7th nerve. The tongue when protruded deviated slightly to the left of the median line; it showed slight fibrillary tremor. Sensation was intact on the face, head and neck. The vision and hearing were good. There was no stiffness of the neck.

Both upper extremities were weak, the right considerably more than the left. In the distal portions of the limbs the weakness amounted to almost complete paralysis on the right side, especially for movements of the hand and fingers. Atrophy was very marked in the forearms and in the hands, involving the thenar and hypothenar eminences and interossei muscles. This atrophy was more marked on the right. Some contracture and deformity was present in accordance with the paralysis and atrophy. Relatively, power was largely retained above the elbows. Touch and pain senses were retained but diminished in the upper extremities. The patient apparently failed to locate the pain. Temperature appeared normal in the upper arms but was markedly affected in the forearms, most notably on the ulnar side, and was lost in the right hand. The same conditions were present but not so positive on the left side. Sense of position was retained on each side, but the patient could not recognize objects properly, in the main because of loss of power. Some objects could be recognized in the partially paralyzed left hand. These notes as to sensation were taken when she was first examined. Later hyperesthesia appeared in the entire left upper extremity.

The tendon and muscle jerks in the upper extremities showed some increase and variation.

Wasting was general of the muscles of the trunk, especially of those of the back. The patient was unable during the eight weeks of her stay to sit up in bed without support, apparently because of the weakness of the trunkal muscles.

The sensory investigations showed differences at different examinations. In one examination, for instance, impairment of tactile sense was present up to the mammary region. Pain and temperature sensations were impaired to a level below that of the tactile diminution. A late examination for sensation made by Dr. Mills on June 10th gave the following results.

On the ulnar aspect of the hand and forearm the patient called cold, hot; rarely hot, cold. The temperature sensation for cold was somewhat delayed. The areas for impairment of temperature sensation were not definitely outlined, but the preponderance of change seemed to be on the ulnar and postaxial aspects of the hand and forearm. Examination of the upper thorax on the left showed a confusion of hot and cold sensations. The pain sense at this investigation did not appear to be changed, like that of temperature.

Voluntary power in the lower extremities on several examinations was almost gone. The patient could not draw up the limbs while in bed, but flexion and extension of the foot were preserved on each side. No wasting of the lower extremities was detectable. Touch and pain senses were not impaired in the lower extremities, but temperature sense was much disturbed, and so irregularly that the areas of disturbance could not be definitely outlined.

At an early examination after admission to the hospital the knee-jerks were very prompt. The Babinski reflex was present on each side. Slight foot clonus could be elicited on the right side but only at times, while on the left it was not present. A few days later the knee-jerks and ankle-jerks were about normal and no foot clonus or even the extensor Babinski response could be elicited. Two days after this the deep and superficial reflexes were slightly exaggerated. Persistent foot clonus and the Babinski extension of the toes were present on both sides; there was also a tendency to lateral clonus. The von Bechterew, Sinkler and Oppenheim reflexes were all obtained.

The extraordinary variability in symptoms, in both the upper and lower extremities, especially in the motor and reflex symptomatology in the lower extremities, was a striking feature in the case up to a few days before operation. The reflexes and sensation were varied as above indicated. At one examination the patient was unable to move her hands on her wrists, or to draw up either lower extremity; at another she had considerable power in the flexors and extensors at the wrist and could forcibly draw up the lower limbs.

The patient had complete loss of control over the bladder and rectum, both when lying down and when sitting up supported.

As some of the symptoms indicated the possibility of cervical ribs, these were examined for and it was believed were detected by deep palpation. A Roentgen-ray examination was then made by Dr. Henry K. Pancoast, with the following results:

Supernumerary ribs turned downward were found on each side. These ribs were rather rudimentary, the one on the left side being somewhat longer.

The usual vascular symptoms of cervical ribs were not much in evidence. The radial artery on each side was patulous and the pulse well defined on elevating the arms. The pulse volume diminished somewhat when the arm was drawn fully backward.

The etiology of this case may be regarded as uncertain. Although the patient's husband had a specific history, she had not had any miscarriages, and evidences of infection were lacking. Her mother died of tuberculosis, as did also one sister. Another sister died of cancer of the uterus, the father of some form of stomach trouble.

As the patient had been thoroughly treated with iodides and mercury without any improvement, and as the Wassermann and Noguchi tests, made by Dr. E. P. Corson-White were negative, the question of operation was taken up and it was finally decided to have a cervical laminectomy performed for the purpose of exploration, and also for the removal of a growth or of the thickened membranes. The operation was performed by Dr. Charles H. Frazier, June 20, 1910. The following are the notes of the operation:

Under nitrous oxide, ether anesthesia, preceded by morphine grain $1/6$, atropine grain $1/150$, patient lying upon her left side, an incision was made having the 7th cervical vertebra as its center. The spines and laminæ were removed from the 5th, 6th and 7th cervical vertebræ. Upon exposing the dura it was found to be much thicker than normal with dense adhesions between the dura and pia, but not between the dura and bone. The pia also was found to be thickened and there were dense adhesions between the pia and cord. It was quite evident from the appearance of the cord that we were dealing with an inflammatory lesion and not a neoplasm, and that the lesion was of such character as not to be amenable to surgical therapy. Consequently after these exploratory measures we proceeded to close the wound. The pia was closed with fine catgut suture but in order to afford some relief of pressure the dura was left unsewed. The muscles and fascia were sutured with a continuous catgut, and the skin with a continuous silk suture. A small cigarette drain was inserted through a stab wound near the lower angle of the wound. The patient left the operating table in excellent condition but shortly afterwards her temperature began to rise rapidly and her pulse to become more frequent, until within a few hours her temperature was over 104° and her pulse 130.

The patient's condition, however, gradually improved up to a certain point. The wound healed by first intention throughout, and the sutures were removed six days after the operation. Eight days later she was transferred again to the service of Dr. Mills.

The patient died a few days later.

The only necropsy allowed was of the spinal cord and its membranes. These were removed and were examined in the laboratory of neuropathology of the University of Pennsylvania by Dr. Williams. The following is a summary of the results of his examinations:

Macroscopically the dura and pia-arachnoid were very much thickened throughout the entire length. The area of greatest thickening extended from the highest cervical to about the second thoracic segment. The entire cervical enlargement was distorted and deformed, being flattened antero-posteriorly, the membranes here forming a solid mass, firmly adherent to the cord substance. Below this area the inner membranes were firmly adherent to the cord but the dura did not adhere as in the upper segments. Along the posterior portion of the cord in the lower cervical region was slight evidence of an old sub-dural hemorrhage, giving the appearance of a very narrow yellow streak on the pia-arachnoid.

Microscopically the thickening of the membranes was confirmed. The vessels were all very much congested and the changes were in detail as follows:

First Cervical.—The pia-arachnoid and particularly the dura are greatly thickened and the vessels congested. The shape of the section is somewhat irregular. A distinct zone of peripheral degeneration with some degeneration more or less diffuse throughout the section is present. The posterior columns, more markedly the columns of Goll, are so degenerated as to give a condition similar to that of tabes. Degeneration is more intense immediately surrounding the blood vessels and along the septæ of the white substance, these being considerably thickened throughout. The posterior root entering fibers are partly degenerated and none of them appear entirely normal, having a wavy, tortuous, and swollen appearance. The vessel coats are greatly thickened and their luminæ very narrow. The congestion is great in both white and gray substance, especially in the latter, and in some places are perivascular hemorrhages, and some slight degree of vascular infiltration is present. The cells are for the most part normal, though different stages of degeneration can be seen in some of them.

Second Cervical.—The meninges here are much more thickened and a rather intense grade of infiltration can be seen. This is more marked posteriorly and is probably a post-operative reaction. The vascular congestion is only slight. Layers and clumps of newly formed connective tissue cells can be seen throughout the pia. These are particularly numerous about the thickened arteries. This is probably a reactive increase, and its intensity seems to depend in large measure upon the degree of closure of the vessels. Both the anterior and posterior roots contain only a few healthy fibers. The entering root zones show an entire absence of normal fibers, those fibers present being broken down and finely beaded. The anterior roots are divided into columns much more distinctly than normal by an excessive growth of the interstitial trabeculæ.

Many of these cell groups have linear nuclei of typical connective tissue type, while some show a round or oblong shape, this being due either to a transverse cutting of the nuclei as would occur if the connective tissue cells were in axis of the nuclei, or to their being young and round or oblong in shape and not yet having acquired cylindrical form.

The calcified vessels, particularly where the lumen is completely occluded, have the appearance of the arenaceous bodies described by Spiller and lend further proof to his suggestion concerning their origin. The intra-medullary vessels are congested and tortuous and the coats greatly thickened. The ganglion cells are only in a few instances degenerated sufficiently to show any change in structure.

Third Cervical.—The sections here are irregular in shape. The cord is partially twisted and rotated toward the left and flattened so as to form an irregular triangle, the base being formed by the ventral cord surface and the apex consisting of the right posterior horn area. The membranes are thickened, infiltrated, and firmly adherent to the periphery of the cord. The greatly thickened vessels are congested and calcified, the latter condition causing varying degrees of occlusion of the lumen. The posterior roots are greatly degenerated, particularly on the left, but it is doubtful whether any entirely normal fibers are present in the posterior or anterior roots on either side. A broad and distinct zone of peripheral degeneration can be seen and this degenerative change extends diffusely throughout the entire white substance, preponderating, however, in the white matter of the posterior columns. The vessels in the cord are congested and tortuous. The anterior horn cells are slightly changed, but a great many are almost normal. The posterior horn cells, however, are practically absent.

Fourth Cervical.—The entire section is irregularly flattened antero-posteriorly, and the anterior horns spread lateralwards. The extreme thickening of the dura and pia-arachnoid forms a mass strongly adherent to the cord and portions extend as trabeculæ into the substance of the posterior roots. The marked peripheral degeneration has extended also along the septa and is particularly noticeable in the posterior columns and along the borders of the anterior fissure, Gowers' antero-lateral area, and the crossed pyramidal tracts. With the exception of the increased thickness and distorting effect, and the greater number of calcareous arteries (mostly in the postero-lateral portion), the meninges do not differ appreciably from the condition in other levels of the cord. The posterior horn cells are absent almost entirely. Those of the anterior horns are not greatly decreased in number, but many of them are greatly degenerated, and it is doubtful whether any of them are really normal cells. The nuclei in many instances are absent or they are indistinct and irregular in shape, and the cell pigment is increased far in excess of the normal. The vessels are extremely tortuous, congested, and sclerosed, and remains of minute perivascular hemorrhages can be found in the gray substance.

Mid-dorsal.—The sections have the irregular outline of a normal cord. The membranes are not so thickened as in the previously described areas, but otherwise are the same. The cord substance in addition to the zonal area of degeneration, shows degenerative changes extending into the substance, along the septa and the crossed pyramidal tracts, and those in the region of what is known as Gowers' area are almost totally destroyed. Cells are present in Clarke's columns and in both the anterior and the posterior horns, but only in a few in the posterior horn was there an approach to the normal condition, and none even of these could be called healthy functioning cells. Numerous amyloid bodies are seen throughout the posterior columns and in lesser numbers in the remainder of the section. The degenerated areas are replaced by an overgrowth of sclerotic tissue of a connective tissue character.

Second Lumbar.—The meninges are not so thickened nor so infiltrated as in the foregoing levels and the cord has a regular contour. The anterior roots are slightly degenerated, the posterior ones much more so; in these latter the fibers are very beaded in appearance. In both the anterior and the posterior roots the overgrowth of connective tissue trabeculæ is excessive, and the vessels are congested. The area of peripheral degeneration is not so broad as in the other levels. The changes along the septa are only slight. The most extensive degeneration is in the crossed pyramidal region. The posterior horn cells are almost absent, and those present are degenerated. The cells of the anterior horns are numerous and most of them are only slightly changed.

Correlating in summary form the gross and microscopical findings, especially the latter, with the symptomatology of this case the following conclusions would seem to be justified.

In the first place, of interest microscopically would be the diffuse and extensive character of the meningitis, with the preponderance of the changes in the cervical enlargement. The infiltration was more excessive in the cervical region and particularly in the posterior part. This was probably due to postoperative reaction, but might have been confused with a possible luetic process. The degeneration involving so many of the fibers of the posterior columns, as well as those of the antero-lateral tracts, seems to have been greatly out of proportion to the minor degree of objective sensory changes. The excessive degeneration of the posterior roots and of the posterior horn cells throughout the cord would also lead one to expect greater sensory involvement. The greater involvement of the temperature and pain senses as shown in the clinical history of the case may find its explanation in the location of these areas of degeneration.

Degeneration of the anterior horn cells became very marked, first at and below the level of the fourth cervical segment, this explaining the greater loss of power and atrophy in the forearms and hands. The wasting and weakness of all the trunkal muscles were also attributable to the degeneration of the anterior horn cells in the dorsal region. The cellular constituents of the lumbar anterior horns being so well preserved accounted for the absence of any atrophy in the lower extremities, whereas the loss of power and the increase of deep reflexes were dependent upon the distinct degeneration in the cross pyramidal tracts. The vascular thickening and tortuosity of the vessels with calcification of their coats in many instances, and the extension of the degeneration from the immediate vicinity of these vessels and the trabeculæ together with the overgrowth of the latter in both the cord and the root substance are of some interest.

The various degrees of closure of the luminæ of the calcified vessels caused different degrees of increase of the surrounding connective tissue cells. The vessels which were completely closed having the appearance of corpora arenacea and thus gave more evidence to the theory of the vascular origin of these bodies. The variability of the clinical symptomatology might be accounted for by vascular conditions. When vessels are so sclerosed and their coats so thickened as in this case, it would not require great systematic vascular changes to cause marked changes in the cord, such as anemia or congestion. On the other hand where there are so few normal cells present, these more readily become fatigued in such a way possibly as to cause this great variation in the symptoms.

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THE SENSORY TRACT IN RELATION TO THE INNER CAPSULE.[1]

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Having obtained four years ago the material from a second case which has permitted us to draw conclusions regarding the position of the central sensory tract in relation to the inner capsule, it has seemed desirable to return to a discussion of this subject.

One of us, in association with Dr. F. X. Dercum,[2] in 1902, reported a case which afforded an opportunity for investigation regarding the position of the sensory fibers in a part of their course from the thalamus to the cerebral cortex. A man, aged 49 years, about 1892 became aphasic and paralyzed suddenly on the right side for both motion and sensation. He recovered his speech in about two months, and motion in the upper and lower limbs in about three months. Sensation, however, never returned in the right side. In 1899 there was spastic hemiplegia of the right side, moderate in severity, with a moderate degree of contracture and spastic rigidity of the right upper limb, and exaggeration of the right patellar reflex. The hemianesthesia was well defined by the middle line of the body, and appeared to be complete everywhere on the right side. Right homonymous lateral hemianopsia was associated with the hemianesthesia. The hemianesthesia was studied repeatedly, and while intense it presented the following peculiarities: It was found that upon several occasions the patient would respond when the trunk upon the hemianesthetic side was tested, or when the proximal parts of the limbs were tested, that is, the upper arm and shoulder or thigh and hip. It was noticed also that occasionally he would respond when the right side of the face below the eye and above the mouth was touched. At most of the examinations no responses were obtained in these situations. At different times also it was noted that the line defining the hemianesthetic from the side with normal sensation varied somewhat, at times retreating a little from, and at other times advancing toward, the sound side. In the distal portions of the limbs, such as the forearms and hands and legs and feet, no variations in the tests were ever observed. The hemianesthesia appeared to involve equally all the forms of cutaneous sensation. The patient died January 9, 1901.

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Read at the thirty-seventh annual meeting of the American Neurological Association, May 11, 12 and 13, 1911.

[2] Dercum and Spiller, American Journal of the Medical Sciences, March, 1902.

The left basal ganglia were cut in microscopical serial sections. A cyst implicated the posterior portion of the lenticular nucleus, the outer part of the posterior portion of the posterior limb of the inner capsule, the area of the *carrefour sensitif,* and the optic radiations. The greater part of the posterior limb of the internal capsule was intact. The optic thalamus was not implicated at any part in the cyst. The cyst was limited in its outer side in its upper portion by the cortex of the insula. The posterior part of the optic thalamus in its superior portion contained few nerve fibers, because of the secondary degeneration from the lesion of the optic radiations, but at a little lower level more fibers were found entering the posterior part of the optic thalamus. The cyst had the same relative position in all the sections as far as the lower part of the temporal lobe. In sections from lower levels the posterior part of the posterior limb of the internal capsule was even less implicated than in those from higher levels.

This case seemed to be one of great importance. The hemianesthesia was such as has been observed after cerebral lesions, and persisted probably over eight years. It was much more intense than the hemiparesis, and the reason for this was found in the slight degeneration of the motor part of the posterior limb of the internal capsule. The escape of the optic thalamus in the primary lesion is noteworthy.

The conclusions drawn may be repeated: "This case seems to show that organic hemianesthesia may be caused by a lesion in the *carrefour sensitif* and lenticular nucleus, without implication of the optic thalamus, except such as occurs from secondary degeneration. We emphasize the statement that in this case the inferior and external portion of the thalamus was intact. It seems to be the first case of the kind carefully studied in the literature. Whether or not the implication of the lenticular nucleus is necessary for the existence of organic hemianesthesia we can not determine by a study of our specimens. The integrity of almost the whole of the posterior limb of the internal capsule seems to indicate that the sensory fibers are located chiefly, if not entirely, in the area of the *carrefour sensitif*, or it may be that some sensory fibers pass through the lenticular nucleus. This view is in accord with the teaching of Edinger, inasmuch as he states that a portion of the sensory tract passes through the posterior third of the internal capsule and a portion through the lenticular nucleus. The lemniscus in its interolivary portion on the left side was one-fourth to one-fifth narrower than that on the right side, and from this we conclude that the lemniscus on the left side had undergone retrograde atrophy. Some of its fibers, therefore, were probably cut in the lesion of the *carrefour sensitif* and lenticular nucleus."

The atrophy of the left lemniscus in this case as well as in the second case reported in the present paper, is interesting. It seems now well established that no fibers of this tract pass to the cerebral cortex, but that all terminate in the optic thalamus. Inasmuch as none of these fibers could have been cut, if this view be correct, by the cyst in either of these two cases, the lesion of the thalamo-cortical sensory fibers must have caused retrograde atrophy in the lower system of sensory neurones in association with the thalamo-cortical sensory neurones.

The various theories relating to the function of the optic thalamus were referred to in this paper by Dercum and Spiller, and it is neither necessary nor desirable to repeat what was said there concerning those that time has shown must be discarded. The views of Dejerine,[2, 3] either as given by himself alone or in association with Long, are so important that mention should be made of them again. It is not possible, according to Dejerine, to

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[2] Dejerine, Anatomie des Centres Nerveux, Tome deuxième, fascicule I, p. 257.

[3] Dejerine and Long, Comptes rendus de la Soc. de Biologie, 1898, p. 1174.

admit the existence in the posterior segment of the internal capsule of a region for sensory fibers only. The sensory fibers are intimately mingled in the internal capsule with the fibers of projection. Hemianesthesia of general sensation, of central origin, can exist only when a lesion in the optic thalamus destroys the terminal fibers of the median lemniscus and the thalamo-cortical fibers; or when the connections of the thalamus with the sensori-motor cortex are destroyed, even though the thalamus may be intact; and in the latter case the lesion is always very extensive.

Long,[4] in his thesis, says: The sensory tract passes through the tegmentum of the cerebral peduncle and enters the inferior and external part of the optic thalamus, from this the thalamo-cortical fibers pass by the internal capsule and corona radiata. Hemianesthesia occurs:

In the cases in which the optic thalamus being intact the connections with the sensori-motor cortex are more or less interrupted, in the latter condition the lesion is always very extensive. Hemianesthesia is especially likely to be persistent when the lesion is in the optic thalamus (pp. 105, 106).

A distinct sensory tract in the posterior segment of the inner capsule does not exist, the corticopetal fibers are mingled with the other vertical or transverse fibers, and especially with the fibers of the pyramidal tract, which occupies the knee and the posterior segment of the internal capsule as far as the retrolenticular region (p. 93):

Marie and Guillain[5] do not believe in the existence of a distinct sensory tract in the internal capsule. They say: "To the observations which we have mentioned above, and which prove that lesions of the sensory zone of the internal capsule may exist without anesthesia, we can add more than thirty cases in which we have seen degeneration of different parts of the lenticulo-optic segment without hemianesthesia.

"We believe that we have shown in this work that it is not possible to describe a region exclusively sensory in the inner capsule of man, and that hemianesthesia may not occur even with intense capsular and cortical lesions.

"One may observe destructive lesions of the so-called sensory zone of the internal capsule without persistent hemianesthesia.

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[4] Long, Les Voies Centrales de la Sensibilité Générale, 1899.

[5] Marie and Guillain, Semaine Médicale, 1902.

We do not know whether the sensory fibers pass through the part of the inner capsule designated by Türck and Charcot, but we may assert that when this zone is damaged sensation may follow other tracts to reach the field of consciousness, consequently tracts confined wholly to the transmission of sensory impressions do not exist in the inner capsule."

It appears from this that Marie and Guillain do not exclude the existence of sensory fibers in the internal capsule; they seem to grant that the *carrefour sensitif* may contain sensory fibers, but they add: "We have been led to ask ourselves whether sensation may not employ many ways of transmission, and an affirmative reply seems to be proper for this question."

Roussy[6] referring to work done by Dejerine, chiefly in collaboration with Madame Dejerine or Long, states that the motor and sensory tracts, intermingled in great measure in their cortical distribution (sensori-motor zone), are intermingled in their subcortical and central regions (corona radiata and internal capsule). The motor tract passes directly from the internal capsule into the foot of the peduncle, while the ascending fibers of the sensory tract, coming from the tegmentum, are interrupted in the optic thalamus, which they enter in the ventral and posterior portion. It is at this level that a destructive lesion may cut the sensory fibers and merely graze the projection fibers of the motor path, and it is exactly at this region the lesion occurred in the three cases of the thalamic syndrome that he had been able to study in serial sections (p. 338).

Roussy adds that Dejerine and Long have shown in a communication concerning the localization of capsular hemianesthesia, that disturbance of general sensation results from central lesions of the hemispheres under two conditions:

1. In thalamic lesion destroying the terminal fibers of the sensory tract of the peduncle and the fibers of origin of the thalamo-cortical neurones.

2. When the thalamus is intact but the connections with the sensori-motor cortex are more or less destroyed. The lesion then is always very extensive.

When the lesion is situated in the external nucleus of the thalamus, implicates more or less the internal and median nuclei and the pulvinar, and only a part of the fibers of the posterior segment of the internal capsule, the clinical picture of the thalamic syndrome is seen.

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[6] Roussy, La Couche Optique. G. Steinheil, Paris, 1907.

Such a lesion cuts the centripetal ascending neurones, the central tracts of general sensation which have been interrupted in the thalamus, but he is not able to go further and to say exactly where these different tracts are situated, as he believes with Long, that the median fillet represents only an important part of the sensory paths (pp. 339, 340).

In a footnote Roussy adds: From the recent work of Grünbaum and Sherrington, and Campbell, it seems that the opinion regarding a sensori-motor cortex should be reconsidered, and the ascending frontal convolution be regarded as purely motor, and the ascending parietal as sensory. These most interesting observations, however, have not been confirmed by the study of focal cerebral lesions (338).

In Roussy's case Kaiser, Case 4, a lesion of the thalamus caused degeneration which he was able to trace by the Marchi method into the ascending frontal and ascending parietal convolutions. He states that he is the first to trace thalamo-cortical fibers in man by the method of Marchi from the optic thalamus to their termination in the cortex, in a case in which the lesion implicated the thalamus and respected the posterior segment of the internal capsule. Probst however has done this.

From the footnote referred to above, it is evident that Roussy is in some doubt as to the unqualified acceptance of a sensori-motor cortex, at least he indicates that further observation may exclude such acceptance, and that past work has not established the correctness of a sensori-motor cortex.

It is evident that while he accepts the teaching of Dejerine and Long, and believes the sensory and motor fibers are intermingled in the inner capsule (i. e., the thalamo-cortical neurones of the sensory tracts), he is not prepared to say definitely where these tracts leave the ventral and posterior part of the thalamus to enter the internal capsule. He distinctly states that all the different sensory thalamo-cortical tracts are not known, and that they are more than the continuation of the median fillet.

It is interesting to observe that in his summary of the parts which must be affected to cause the thalamic syndrome he includes a part of the posterior segment of the internal capsule ("et n'intéresse qu'une partie des fibres du segment postérieur de la capsule interne"). An examination of the drawings accompanying his cases permits the conclusion that a lesion of the retro-lenticular portion of the posterior limb of the internal capsule was important in the production of the syndrome, although in Case 4 the retro-lenticular portion escaped.

V. Monakow[7] says that in recent years a number of cases occurring in early life have been published, in which notwithstanding complete interruption of the posterior limb of the internal capsule the anesthesia was not complete, or was only transitory (Dejerine, Marie and Guillain). According to these writers a hemianesthesia may occur from a lesion of the internal capsule, but all the thalamo-cortical fibers, mingled with the motor cortico-spinal tract and covering a large area, must be implicated. Hemiplegia is always associated with the hemianesthesia produced by such a lesion. Marie and Guillain go further, and not only with Dejerine deny the existence of a sharply defined sensory tract in the internal capsule (the *carrefour sensitif*), but from their clinical observations they conclude that destruction of the entire inner capsule, the Rolandic zone, the lenticular nucleus and the optic thalamus, may occur without necessarily causing hemianesthesia. V. Monakow, in reply to this statement, calls attention to the failure of Marie and Guillain to mention whether all forms of sensation, especially the stereognostic perception, were thoroughly studied.

He adds: "It is certain that in the majority of cases of lesion of the posterior part of the internal capsule, and even when the ventral thalamic region is also damaged in extensive degree, the sensations of pain and pressure, although usually in modified form, may be preserved. The same is true of the sensation of temperature, which usually is only partially affected. It is otherwise as regards the muscular sense, the senses of location, space, strength, and stereognosis.

"The muscular sense, when the entire retro-lenticular portion is destroyed, can scarcely ever be entirely intact. The same is true of location and space senses. These forms of sensation, as the majority of clinical cases observed to this time and experiments on animals without exception have demonstrated, are permanently greatly impaired by so-called cortical lesions, i. e., those confined to the Rolandic region. As regards the behavior of the stereognostic sense, I have never missed a serious disturbance of this sense in the cases of lesion of the retro-lenticular inner capsule observed by me. If indeed the stereognostic sense in the cases of Dejerine and Long, and Marie and Guillain (notwithstanding the extensive capsular lesions) has been entirely intact, which is incomprehensible to me, this negative finding must be explained by the long duration of the lesion and by the assumption of the previously lost functions by the sound hemisphere (substitution)."

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[7] v. Monakow, Gehirnpathologie, second edition, p. 611.

V. Bechterew[8] concludes from his own clinical observations, that extensive destruction of the anterior and medial portions of the thalamus may exist without hemianesthesia. Only lesions of the posterior thalamus are constantly associated with anesthesia of the contra-lateral side of the body. One must not conclude, he says, that the hemianesthesia which occurs with the so-called capsular lesion is dependent in all cases on a lesion of the thalamus. As the sensory tract from the thalamus to the cerebral cortex passes through the internal capsule, evidently a focal lesion of the upper part of the capsule may cause hemianesthesia or hemiplegia. Accordingly experimental and clinical observations are in evidence that the posterior outer part of the thalamus, or more correctly, the posterior part of its lateral nucleus, forms a station for the sensory tracts from the contralateral side of the body.

G. d'Abundo[9] regards the term thalamic syndrome as badly chosen, inasmuch as the posterior segment of the internal capsule was implicated in all the cases presenting this syndrome in which the thalamus was affected. He therefore does not accept the view that the optic thalamus is a station in the sensory tracts. In his experimental work he found slight and transitory impairment of general sensation as a result of lesion of the postero-external portion of the thalamus, and marked impairment of sensation occurred only when the internal capsule was implicated.

That a lesion in the upper and external part of the optic thalamus may not produce hemianesthesia is shown by the well-known case of Edinger,[10] reported some years ago. His patient did not have hemianesthesia. At first following the onset of the symptoms, he states she had a sensation in the right side different from that in the left. She had distinct hyperesthesia in the paralyzed limbs. Tactile impressions, unless they were very slight, were felt as pain. The lesion occupied the dorsal part of the external nucleus of the optic thalamus and a part of the pulvinar.

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[8] v. Bechterew, Die Funktionen der Nervencentra, 1909, Vol. 2, p. 1112.

[9] G. d'Abundo, Abstract in Revue Neurologique, Dec. 15, 1910.

[10] Edinger, Deutsche Zeitschrift für Nervenheilkunde, Vol. I, p. 262.

These words are found in Lewandowsky's[11] recent work: "The position of the sensory tracts is somewhat more complicated than Charcot assumed in his teaching of the *carrefour sensitif,* but the greater part of this tract is caudal to the motor tract, and without doubt the anatomical possibility is thereby given that at least the chief portion of the sensory fibers may be destroyed by a lesion without implication of the motor tracts. In fact Raymond, Dercum, Spiller, and v. Monakow have observed cases of pure hemianesthesia from lesions of the retro-lenticular portion of the internal capsule, v. Stauffenberg from a lesion in the medullary substance of the posterior central and supramarginal gyri, I also have seen pure hemianesthesia in an operated case, and from the position of the tumor, which was removed, the cause of this persisting anesthesia, even after the operation, was not to be found in a lesion of the thalamus, and not alone in a destruction of the cortex, but must be attributed to a lesion of a part of the inner capsule (Vol. 2, p. 795 et seq.).

The character of the sensory disturbances from thalamic lesions, Lewandowsky says, does not differ in its details from that from a lesion of the internal capsule or of the cerebral cortex (p. 792).

The usual result of lesions which destroy the corona radiata of one side, is not extensive restoration, but persisting disturbance even almost complete loss; and yet complete loss of sensation and especially of pain sensation, scarcely ever occurs even immediately after the apoplexy; according to Marie it never occurs (p. 793).

Inasmuch as sensation of the extremities may be more disturbed than that of the face, and vice versa, from thalamic lesions, the sensation of different regions of the body must be separately represented in the thalamic nuclei, but we do not know exactly what this localization is. We know nothing concerning the localization within the sensory part of the inner capsule. We can not tell from clinical signs whether a disturbance of sensation depends on a lesion of the thalamus, inner capsule or cortex. The predominance of sensory over motor disturbance or its isolated occurrence, indicates a lesion of the thalamus, the posterior part of the internal capsule, or the region posterior to the central fissure (pp. 797, 798).

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[11] Lewandowsky, Handbuch der Neurologie,

It may be wise to quote Lewandowsky's definition of the ventral nucleus: Most authors, he says, who recognize sharp limitations in the optic thalamus describe a dorsal and a ventral part of the lateral nucleus. The ventral part embraces, according to v. Monakow, the entire basal half of the lateral part of the thalamus (Vol. I, Part I, p. 251).

The studies of Probst[12] on the central sensory tract are among the most important in literature. He says:

"I was able to demonstrate by a series of operations cutting tracts and by the Marchi method, that all fillet fibers enter the thalamus, and that not a single fiber goes directly to the cerebrum.

"After destruction of the latero-ventral thalamus nucleus I could demonstrate a series of sensory disturbances, and as following these lesions the thalamo-cortical fibers to the sensory region degenerated, the conclusion is justifiable that this was the central sensory tract. The central sensory tract makes exit in part through the external medullated lamina of the thalamus, into the ventral part of the inner capsule, and passes upward close to the lenticular nucleus; in part through the dorsal part of the third segment of the lenticular nucleus, into the corona radiata, in which it forms chiefly the lateral arciform fibers (Bogenanteil), and divides in the gyrus sigmoideus anterior et posterior, or gyrus centralis anterior et posterior.

"From my findings the anterior central convolution must be regarded chiefly as motor, the posterior central convolution as sensory."

In regard to the mingling of motor and sensory fibers in the internal capsule, he says: "Exner, Flechsig, Henschen, Dejerine and Long, and Munk have advanced the view that the cortical areas in which motor impulses arise, serve also for perception and extension of the sensory impressions, accordingly motor and sensory tracts would intermingle in the corona radiata.

"In opposition to this view I might refer to my representations of the course of the pyramidal tract through the corona radiata, as made by me from the Marchi staining in amyotrophic lateral sclerosis. If one compares the course of the pyramidal tract in the corona radiata given there with the course of the above described central sensory tract, he will find that both tracts in many particulars correspond in the ventral portion of the inner capsule and also in the corona radiata, but in general it may be said that the central sensory tract is more caudal."

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[12] Probst, Sitzungsberichte der kaiserlichen Akademie der Wissenschaften, Vol. CXV, Part III, 1906.

Reviewing these statements of different authors it becomes evident at once that much difference of opinion exists regarding the positions of the sensory and motor tracts in the internal capsule and corona radiata. There is evidence that the sensory fibers may not be mingled with the motor. Lewandowsky holds, as has been stated, that the bulk of the sensory fibers is behind the motor tract, and this view is taught also by Probst. Some work done by Probst, by which it was shown that the muscular and position senses may be altered by a cerebellar lesion, seems to indicate that we by no means know fully the central pathways of sensation. He says: "I and others have demonstrated after destruction of the cerebellum, disturbance of the muscular sense and of the sense of position, from which the conclusion may be drawn that a part of the sensory conduction is through the cerebellum (l. c., p. 158).

The weight of opinion is in favor of the termination of sensory fibers in the ventral portion of the lateral nucleus, but it is due to Probst especially that we have an anatomical knowledge concerning the thalamo-cortical sensory tract. He has shown that this bundle is distinct from the motor, although probably it is not confined to the area of the *carrefour sensitif;* rather it occupies the outer part of the inner capsule close to the lenticular nucleus, but he states quite positively that in relation to motor fibers "die zentrale Fühlbahn kaudaler gelegen ist." In the interpretation of the case of Dercum and Spiller the possible importance of the lenticular nucleus to the sensory tract was recognized, as shown by these words: "It may be that some sensory fibers pass through the lenticular nucleus."

Most investigators, since the publication of the paper by Grünbaum and Sherrington, regard the precentral convolution as chiefly if not entirely motor, and the postcentral convolution as chiefly if not entirely sensory. From à priori reasoning it would seem improbable that the motor and sensory fibers should be mingled intimately in the internal capsule and corona radiata, if their cortical representation is in so large measure distinct.

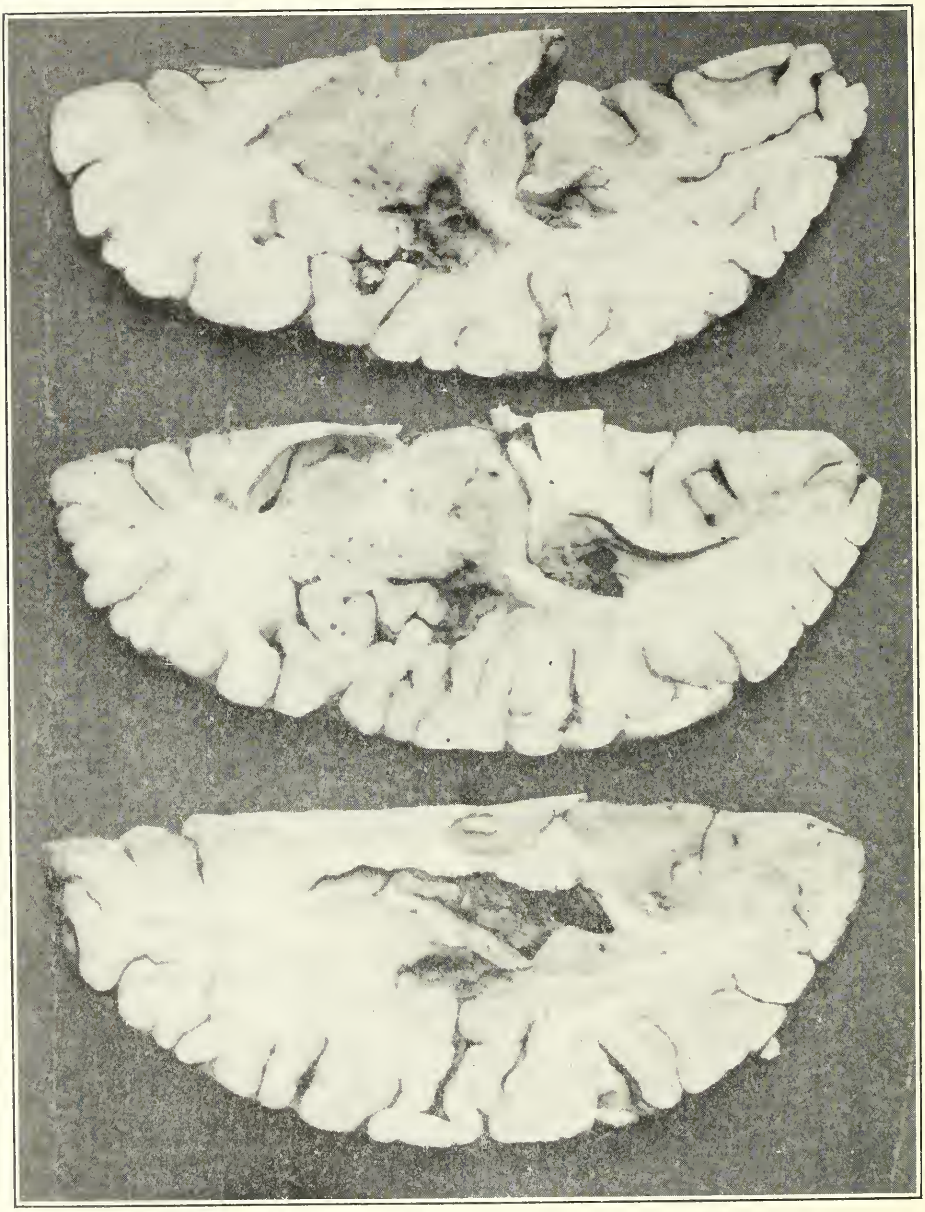

Fig. 1. Photographs of three levels of the left basal ganglia. The lesion destroyed the carrefour sensitif and the posterior part of the lenticular nucleus.

The evidence seems to be clear that a lesion of the ventral portion of the lateral nucleus of the optic thalamus may cause hemianesthesia, but we endeavor in this paper to again demonstrate, what was attempted in the paper by Dercum and Spiller, viz., that a lesion implicating the posterior part of the internal capsule and the posterior part of the lenticular nucleus also may cause hemianesthesia, and that in these two cases the hemianesthesia can not be attributed to a thalamic lesion. It is true that in the present case a very small lesion was found only in the dorsal part, that is in the upper part, of the optic thalamus, but it did not extend to or near to the ventral nucleus, and was situated similarly to the lesion in Edinger's case, which produced no impairment of sensation.

If the objection be made that hemiparesis existed in our cases, and therefore sensory and motor fibers were mingled, our reply would be that hemianesthesia occurs when the posterior part of the internal capsule and the posterior part of the lenticular nucleus are degenerated, and that many cases of hemiplegia without hemianesthesia are known in which the lesion was in the middle or anterior part of these structures.

The case we describe is as follows: The patient was a man who died in the service of Dr. Charles K. Mills immediately before one of us (Dr. Spiller) went on duty, but Dr. Mills has kindly given us the pathological material for study. The man was admitted to the Philadelphia General Hospital October 10, 1900, and died January 1, 1907. A brief reference to this case had been made in a paper by Dr. Weisenburg[13] and we quote his notes:

"W. K, white, married, aged 66 years, cement worker, denied syphilis, but was a moderate drinker, and had been healthy until fifteen weeks previously, when, while working in the street on a hot day, he fell and was unconscious for about twenty hours. On recovery he had a complete right-sided hemiplegia. His speech was not affected.

"He was admitted to the Philadelphia Hospital five weeks after the stroke. When admitted he had regained partial use of the right arm and leg, the leg having improved more than the arm. The arm shows moderate contracture.

"The patient was examined on admission by Drs. Mills and Spiller. The examination showed absence of all forms of sensation over the right arm to a point corresponding to the insertion of the deltoid muscle. From here to the shoulder, sensation was impaired, and still higher it became less marked. At this examination the anesthesia in the lower extremity was also more marked distally. All forms of sensation were absent to an irregular line just below the knee, impaired from this point to the hip, while on the abdomen and chest they were but slightly diminished. The anesthesia in this case was not limited to the median line either on chest or face. In the latter it reached to the side of the nose and a part of the cheek on the other side. Sensation was dulled on

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[13] Weisenburg, Journal of Nervous and Mental Disease, 1901, p. 285.

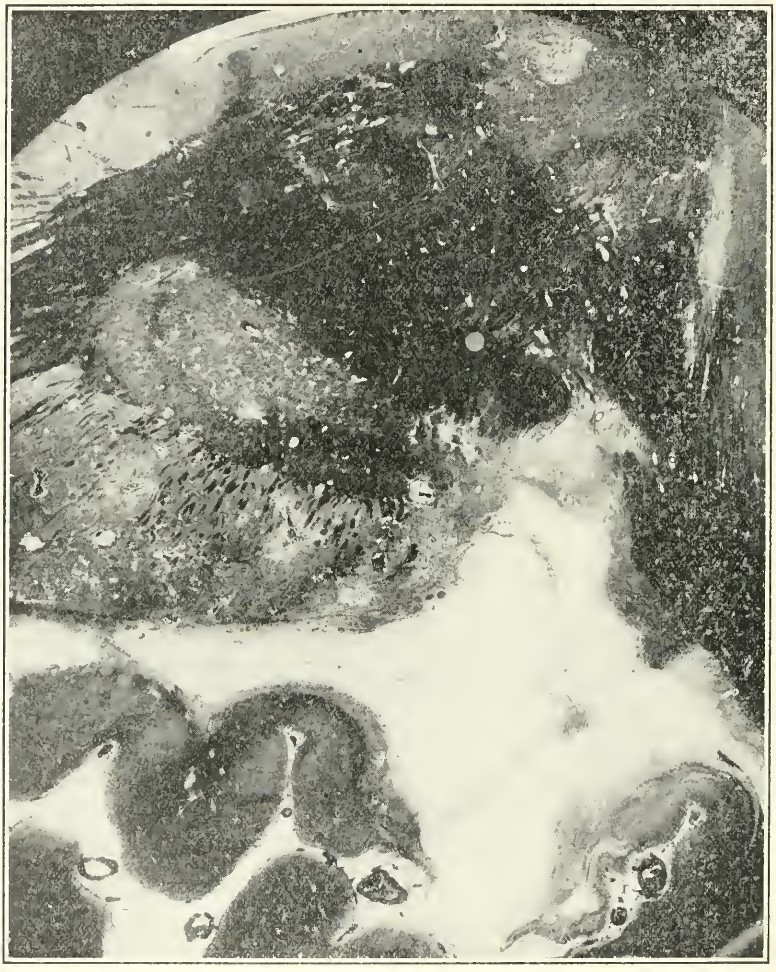

FIG. 2. Photograph of microscopical section taken from the middle block of Fig. 1. (Photograph by Dr. A. J. Smith.)

the right side of the tongue and absent on the palpebral conjunctiva. Muscular and pressure senses were absent, and stereognosis was present. [It is not stated whether stereognosis was impaired or not, presumably it was.]

"Subsequent examinations have never given the exact results above noted. The most frequent results obtained in the later examinations have been entire absence of tactile and temperature sensations for the whole right side; pain sensation, however, being retained in some areas of the shoulder, chest, abdomen and thigh."

This patient was repeatedly in the service of one of us during the years he remained at the hospital, as well as in the service of other members of the staff. Such data as relate especially to the motor and sensory palsies are taken from the later notes, but they are less reliable than those obtained in the examination by Drs. Mills and Spiller.

Nov. 9, 1900. He has total tactile anesthesia on the right side. Pain and thermal sensations are impaired, and muscular sense is lost on the right side.

Dr. de Schweinitz, at this time, recorded the presence of incipient cataract in the right eye. He obtained a hazy view of the left retina. Hemianopsia was not present.

Nov. 14, 1901. There is partial loss of power in the right arm and leg, but the man walks about at will.

Jan. 6, 1903. Grip is fairly good on each side, is slightly diminished on the right side. Slight contracture is present in the right upper limb. Power in the right lower limb is diminished. Babinski's reflex gives upward movement of the toes on the right side, but there is no ankle clonus.

Anesthesia is present over the right side of the head, scalp and face, extending 2 to 3 cm. to the left of the median line. On the chest and abdomen the anesthesia extends irregularly 1 to 2 cm. beyond the median line. The right upper and lower limbs are anesthetic, at times there seems to be present an after-sensation.

Dr. Shumway reported Oct. 21, 1903, O. D. vision 5/20; O. S. vision at one meter. O. D. media clear, disk rather pale but not pathologically so, vessels are irregular in caliber particularly the veins (arteriosclerosis), otherwise the fundus is normal. O. S. shows opacity in the capsule of the lens. Homatropine dilatation shows eyegrounds to be that of high myopia with extensive choroiditis in macular region.

April 25, 1905. There is hemianesthesia involving skin and mucous membranes, slightly overlapping the sound side. Later some contraction of the visual fields for red and white was noticed.

Pneumonia developed on Dec. 31, 1906, and death occurred Jan. 1, 1907.

One might wish that certain details regarding sensation had been made more complete, but the uncertainty of obtaining a necropsy in any case in this country interferes greatly with exhaustive clinical study. The facts given are sufficient to show that a grave disturbance of sensation persisted during a period of about six years, but it is extremely probable that in some instances the term "anesthesia" was used when it should have been "hypesthesia."

The brain was cut in horizontal microscopical serial sections. The region of the left basal ganglia was sectioned at three levels. A cyst was found in the outer and posterior part of the lenticular nucleus and island of Reil, implicating about the posterior third of the lenticular nucleus. The posterior third of the posterior limb of the internal capsule was degenerated. A small area of degeneration was found in the portion of the optic thalamus adjacent to this degeneration in the internal capsule, but it was only in sections from the higher levels, was very small, and could have had no connection with the disturbed sensation. The retrolenticular region of the internal capsule was normal, and normally stained fibers passed from it into the pulvinar of the optic thalamus. This condition explains the absence of hemianopsia. The anterior limb of the internal capsule, the knee, and anterior two-thirds of the posterior limb, stained normally. In sections from lower levels the degenerated area withdrew from the region of the optic thalamus toward the island of Reil. The ventral nucleus of the optic thalamus was in no wise implicated.

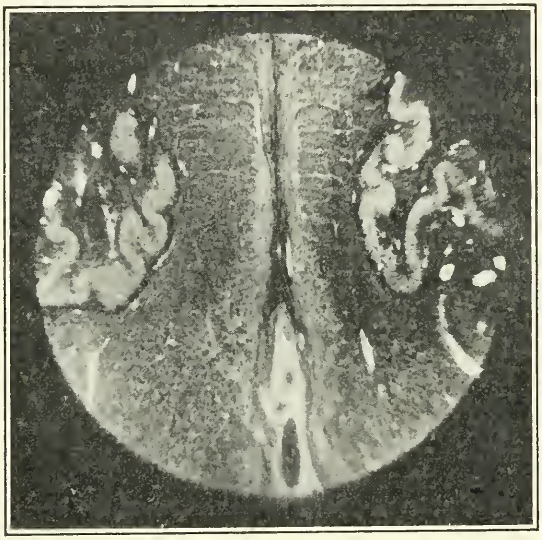

Fig. 3. Photograph of section through the medulla oblongata. The lemniscus on the left side is much smaller than that on the right side. (Photograph by Dr. A. J. Smith.)

The left anterior pyramid was considerably degenerated. The left median lemniscus was about one fourth smaller than the right.

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 39, No. 2, February, 1912.]

PRELIMINARY NOTE ON A NEW SYMPTOM COMPLEX DUE TO LESION OF THE CEREBELLUM AND CEREBELLO-RUBRO-THALAMIC SYSTEM, THE MAIN SYMPTOMS BEING ATAXIA OF THE UPPER AND LOWER EXTREMITIES OF ONE SIDE, AND ON THE OTHER SIDE DEAFNESS, PARALYSIS OF EMOTIONAL EXPRESSION IN THE FACE, AND LOSS OF THE SENSES OF PAIN, HEAT AND COLD OVER THE ENTIRE HALF OF THE BODY[1]

By CHARLES K. MILLS, M.D.

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The clinical history of the case on which this contribution is founded was published in the JOURNAL OF NERVOUS AND MENTAL DISEASE, May, 1908, and also in the Contributions of the Department of Neurology of the University of Pennsylvania for the same year. The present communication is preliminary to a thorough study of the clinical phenomena of the case and of the anatomical, physiological and pathological problems suggested by these phenomena and the findings at the necropsy and microscopical examination. It is my intention to present this more elaborate communication at the next annual meeting of the American Neurological Association, in Boston.

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[1] Presented at the meeting of the Philadelphia Neurological Society, held December 22, 1911. In the presentation of this case before the Philadelphia Neurological Society several lantern slides were shown exhibiting the patient's appearance when the face was at rest, when voluntarily moved and during laughing. Moving pictures were also shown for the writer by Dr. T. H. Weisenburg, of the patient when voluntarily drawing up both sides of his face and showing his teeth and also the absence of movement in laughing. These moving pictures strikingly exhibited the loss of emotional expression on the right side of the face.

The symptom complex is I believe new, never having been described before the publication of the case by me in May, 1908. The entire investigation, clinical and pathological, sheds new light upon the functions of the cerebellum and of the cerebello-rubro-thalamic and the cerebello-rubro-spinal systems. From the point of view of blood supply the syndrome may be regarded as that of occlusion of the superior cerebellar artery.

The patient was admitted to my wards in the Philadelphia General Hospital November 29, 1907, about four years before his death. He was thirty-four years of age, and had a history both of gonorrhea and syphilis.

Ten weeks before admission he was suddenly seized with vertigo, accompanied with nausea and vomiting. He was confined to his bed for about a week. From the time of his seizure he became awkward in the use of his left hand, could not laugh on the right side of his face, although the expression of emotion was the same on both sides previous to the seizure, and he had a sense of numbness or impaired sensation throughout the entire right half of his body—face, trunk, and upper and lower extremities. He was also deaf on the right side, although this fact was omitted by oversight in my report published in 1908.

The symptom complex which this man presented when he was first examined in November, 1907, remained the same until his death on October 31, 1911. During this period he was in and out of the hospital, but his stay in the institution at each period was sufficiently prolonged to give excellent opportunities for the study of his symptomatology.

A summary of an examination made by me October 8, 1911, about three weeks before the patient's death gives the special symptom complex from which this patient suffered for more than four years, this not changing during all that time. Examinations were made for paralysis of ocular muscles, of the seventh and fifth nerve musculature, of the throat, of the limbs, trunk, viscera, etc., with negative results except as will be presently described; in other words the syndrome was clear cut and very definite. In the left upper extremity ataxia was very marked as shown for instance by the finger-to-nose test. As the hand approached the face in the test the movements became very jerky and it was scarcely possible for the patient to touch his nose. In the left lower extremity ataxia was also marked, as shown by the heel-to-knee test. Power in both upper and lower limbs was however preserved. Careful tests showed that sensation in all its forms was fully retained on the entire left half of the body, but was lost to pain, extreme heat and extreme cold on the right half of the body—face, trunk, upper and lower extremities. Tactile discrimination was also greatly impaired as shown by the compass test. Light touch was preserved. The senses of deep pressure and of position and passive movement were normal on both sides and the patient showed no astereognosis in either the right or the left hand. Deafness in the right ear was complete. Voluntary movements in the face were entirely preserved, as demonstrated by such usual tests as wrinkling the forehead, closing the eyes, drawing up the face first to one side and then to the other, and then on both sides as in showing the teeth. In laughing, as had been shown in many other examinations and by the moving picture method, the patient's face failed to move on the right side.[2]

The patient died October 31, 1911. The brain closely examined by the naked eye showed a smallness of the branches of the left superior cerebellar artery and a depression over the left dentate nucleus, but nothing else externally, at least nothing of importance as regards the symptomatology above described. The brain was sectioned in several places with care so as not to interfere with subsequent microscopical investigations by serial sections. The cuts revealed a destructive lesion involving the left dentate nucleus and the cerebellum above this nucleus, including also the superior cerebellar peduncle. Degeneration was also evident to the naked eye in the right nucleus ruber, which body was much smaller than the left nucleus ruber. Many sections of the cerebellum, cerebellar peduncles, pons, crura, etc., have since been made in the laboratory of neuropathology of the University of Pennsylvania, under the supervision of Dr. Wm. G. Spiller, and

[2] In the present preliminary note many details of the general and neurological examination of the patient are omitted, the object being simply to present clearly the symptom complex and its pathological cause.

these show a well marked destructive lesion and secondary degeneration involving not only the dentate nucleus and neighboring cerebellar substance, but also the left superior cerebellar peduncle, to and including the nucleus ruber of the opposite side. A full report of the findings with photographs and other illustrations will be given in the completed article.

Multiple Hemangioperithelioma of the Brain

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MULTIPLE HEMANGIOPERITHELIOMA OF THE BRAIN*

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Endothelioma or perithelioma is not an uncommon tumor within the cranium and usually is solitary. Endothelioma may occur in the form of multiple tumors on the dura, and sometimes as miliary dural growths, as in a case reported by Dercum, Keen and Spiller.[1] Endothelioma within the cranium usually has its origin from the dura and causes atrophy of the underlying brain by pressure, without infiltrating it. In this form it is one of the most benign of intracranial tumors, and yet it may recur if removed, and become most malignant and may infiltrate the bone above it. It is rare to find numerous tumors of an endotheliomatous character within the brain substance; as L. Bruns says: "In the brain substance itself, endotheliomas are very rare." In the form of perithelioma with the rosette-like arrangement of columnar cells about the vessels, it is more common within the brain substance than in the form of chains or columns of cells or groups of concentrically arranged cells.

I have seen multiple perithelioma within the brain substance only in the case reported in this paper, and have found no similar case recorded. The tumors were very numerous in this case, and every tumor was the site of hemorrhage. These hemorrhages varied greatly in size. The largest measured 2 by 3.5 cm., and some were the size of a pea or less. Most of the tumors had a connection with the cortex at one part, but some were entirely within the brain, one for example, the size of a pea, was within the right lenticular nucleus, and is shown in Figure 1. An old area of sclerosis was in the left upper temporal convolutions.

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* Read before the Pathological Society of Philadelphia, Dec. 14, 1911.

[1] Dercum, Keen and Spiller: THE JOURNAL A. M. A., April 28, 1900, p. 1026.

This case illustrates the malignancy that perithelioma may assume, as the tumors were found in the cerebellum and pons as well as in the cerebrum.

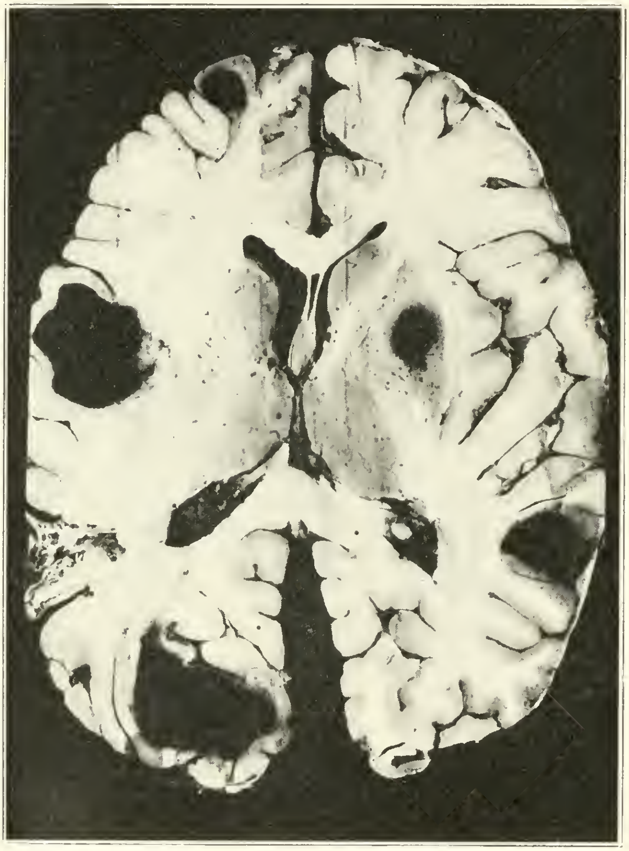

Fig. 1.—Transverse section of the brain in a case of multiple hemangioperithelioma of the brain, showing five tumors with hemorrhage.

It is rare to find more than one recent hemorrhage in a brain, and yet I have at least three specimens in my laboratory in each of which two recent and separate hemorrhages occurred.

Souques[2] reports two remarkable cases of this kind. In one, twenty-eight recent hemorrhages were counted in the brain and cord, and in the other, eleven. Some hemorrhages probably escaped detection on account of the thickness of the sections, and those of the size of a pinhead were not regarded. The hemorrhages were small and mostly just beneath the cortex. Nothing in the clinical condition suggested multiple hemorrhage, any more than in my case. One patient certainly had nephritis, and the other probably had. I do not know of any description of multiple hemorrhage of the brain, each caused by tumor within the brain substance, and for this reason also the case reported here is interesting.

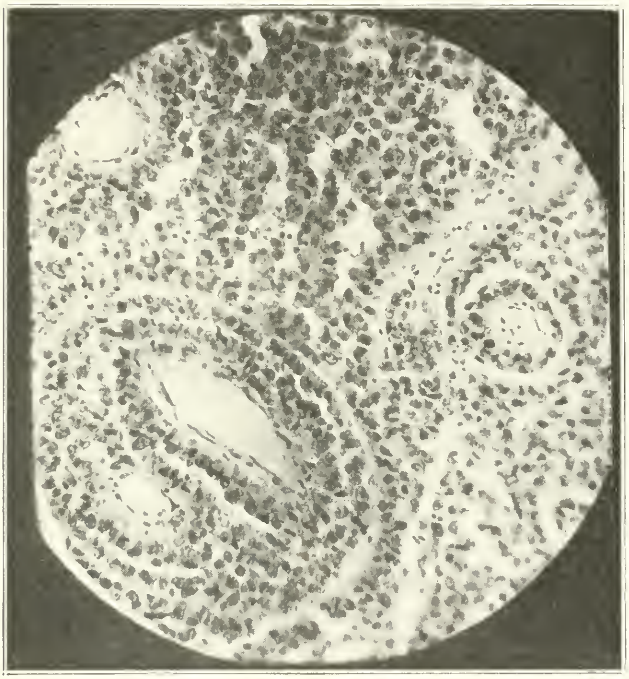

Fig. 2.—Microscopic section of a tumor, from a case of multiple hemangioperithelioma of the brain.

Patient.—C. H., male, aged 60, was referred by Dr. L. B. Heimer to Dr. Mills,[1] who sent the patient to the University

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[2] Souques: Nouv. Iconog. de la Salpêtrière, May-June, 1911, p. 193.

Hospital, Feb. 28, 1911, to be placed under my care. Dr. Heimer furnished the following notes:

History.—During February, 1910, the man suddenly became flushed and reeled when he attempted to stand. His speech was a jargon of syllables and numbers so that he could not be understood, and he did not understand what was said to him. He was unable to walk unassisted; his writing was as much disturbed as his speech. His condition gradually improved, and he returned to work. The sensory aphasia evidently was caused by the area of sclerosis in the upper left temporal convolutions.

In January, 1911, he had convulsions of the entire body, worse in the right side, and the face and eyes were turned to the right. When this attack passed off he was paralyzed on the right side, and remained so a few days.

When he was first seen by Dr. Heimer about the end of 1909, he had a large mass above the sternoclavicular articulation on the left side. Later he had several tumors on each side of his body and in the left elbow joint. The tumors became gradually more numerous and larger. The Wassermann reaction was negative.

Examination.—The man was examined by me March 2, 1911. Paraphasia was marked; he was word-deaf and word-blind. In testing him with a feeding-cup or a bright object, and by striking toward the eyelids, right lateral homonymous hemianopsia was revealed. The right nasolabial fold was not so deep as the left when the face was at rest or when the patient showed the teeth. He could only occasionally be made to show the teeth and then by imitation. The limbs were not paralyzed, but it was impossible to get him to grasp the hand of the examiner firmly. The biceps and triceps reflexes were about normal on the left side, and little exaggerated on the right side. The patellar reflex was about normal on the left side, and exaggerated on the right side. The Achilles reflexes were nearly normal. Upward movement of the toes in the Babinski reflex was obtained on the right side, downward movement on the left side. The right lower limb probably was paretic, as it was not withdrawn with the same vigor as the left when it was stuck with a pin. Numerous tumor-like swellings were found in different parts of the body.

Dr. de Schweinitz examined the eyes and reported: "Right eye, disk shows no choking but upper and inner margins are clouded. Left eye similar, but margins are clearer than those on the other side. No hemorrhages. Pupillary reaction to light present but not very prompt."

Course. A few days after the examination the man developed Jacksonian epilepsy, one convulsion rapidly following the other, implicating the upper part of the face on both sides and the right side of the mouth and the right upper limb. The right lower limb and the left limbs were not affected. After the convulsions were controlled, the right side of the body was in a state of flaccid paralysis. The man did not rally after this attack and died March 13, 1911.

Necropsy. Permission was given for removal only of the brain and cord, and this was done by Dr. Karsner. Numerous areas of hemorrhage within the brain were examined microscopically and perithelioma was found in all the hemorrhages. One of the tumors removed from the trunk was examined by Dr. Karsner and diagnosed as endothelioma.

I am indebted to Dr. Allen J. Smith for the photographs.

1409 Pine Street.

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LOSS OF EMOTIONAL MOVEMENT OF THE FACE WITH PRESERVATION OR SLIGHT IMPAIRMENT OF VOLUNTARY MOVEMENT IN PARTIAL PARALYSIS OF THE FACIAL NERVE.

By William G. Spiller, M.D.,

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The voluntary and emotional movements of the face are independent of one another, and one form may be lost without loss of the other. Von Bechterew[1] says a number of observations show positively that the emotional movements in man are accomplished by means of special tracts which have no relation to the tracts concerned with voluntary movement. Cases of paralysis are known in which voluntary movements of the face, or of the lower part of the face, were lost and emotional movements (laughing, crying) of the face were performed in a normal manner. In discussing these cases, Nothnagel remarked that when the voluntary movement of the face is lost in hemiplegia from a focal lesion, but both sides of the face take part in equal degree in emotional movement, one may assume that the optic thalamus and its connections with the cortex are uninjured. Cases in which the emotional movements of the face have been lost and the voluntary movements preserved have been reported by v. Bechterew, Nothnagel, Rosenbach, Kirilzew, Gowers, and many others.

According to v. Monakow,[2] it has long been known that in many cases of hemiplegia the face on the affected side cannot be moved voluntarily, but the paretic facial muscles at once contract, as well as those of the other side, when the patient laughs or cries. v. Monakow expresses his belief that a part of the sensory reflex tract for laughing or crying is situated in the optic thalamus.

The experimental work of Roussy[3] throws doubt upon the optic thalamus as a reflex centre for emotional movements of the face, as does the work of Ernest Sachs,[4] who, in the report of his experiments upon the optic thalamus states that stimulation produced facial movements of the opposite side. He adds: "It is desirable, perhaps, to repeat here that these motor phenomena constitute no evidence of special centres in the thalamus for automatic or rhythmic movements, as has been assumed by some authors."

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[1] Die Funktionen der Nervencentra, vol. ii, p. 1144 et seq.

[2] Gehirnpathologie, Nothnagel's System.

[3] La couche optique, p. 185.

[4] Brain, Part 126, vol. xxxii, p. 1909.

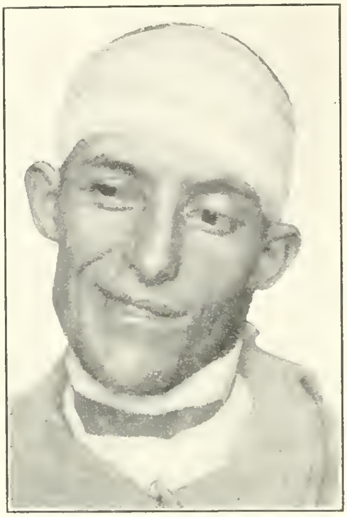

Fig. 1.—In smiling, only the right side of the face showed contractions of muscles.

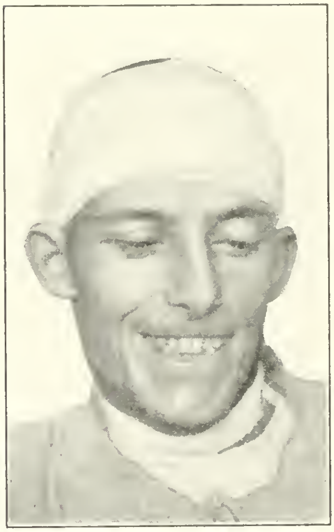

Fig. 2.—In showing the teeth, contractions of the muscles were only a little less pronounced on the left side of the face. The reproduction does not show these contractions so well as does the photograph. The photographs were taken after the operation. The dissociation of facial movements was more pronounced before the operation.

One possibly might make the objection that the results obtained in animals are not applicable to man.

Lewandowsky[5] believes the opinion regarding the optic thalamus as an emotional centre is not well founded. There are so many negative cases in the literature that a relation of paralysis of emotional movement to thalamic lesions cannot be maintained. Even in the cases in which thalamic lesions have been found, different parts of the thalamus have been affected. It is not improbable, he adds, that the assumption of a relation between emotional facial movements and a thalamic lesion is without foundation.

The paralysis of one form of facial movement without impairment or with only slight impairment of the other may result from a lesion of the facial nerve, or may be a pseudoparalysis from imperfect innervation. I have observed persons who in smiling or speaking innervated one side of the face so much less than the other that paresis of one side seemed to be present, and yet the drawing up of the corners of the mouth, as in showing the teeth, was equally good on the two sides.

What I especially desire to call attention to at the present time is loss of emotional movement of one side of the face with preservation or possibly only slight impairment of voluntary movement of the same side, as indicative of a certain stage of peripheral facial palsy. I have observed this phenomenon especially in slight facial weakness resulting from the pressure of a tumor of the cerebellopontile angle upon the facial nerve. The movement of the face normally is less intense in smiling or crying than in showing the teeth, and, therefore, a slight paresis of the facial nerve might cause loss of these emotional movements on one side of the face, and the weakness might be overcome by the forcible effort made to contract the lower part of the face in showing the teeth. The case was one referred to me by Dr. W. H. Snyder and Dr. Louis Miller, of Toledo, Ohio, October 23, 1910. In smiling the man drew up the right side of the face strongly and the left side weakly, but in showing the teeth or drawing up the corners of the mouth separately, the left facial weakness was slight. Dr. C. H. Frazier operated and found a tumor in the left cerebellopontile angle pressing upon the facial nerve.

It is important to recognize that this form of dissociation of facial movement may be the first sign of weakness of the facial nerve from pressure of a cerebellopontile angle tumor, and in connection with slight nerve deafness may be of localizing value. It is possible that the impairment of emotional movement may result, in part at least, from the pressure of the tumor upon the medulla oblongata and pons. I am unable to determine at present whether or not the impairment of emotional movement of the face occurs in most cases of tumor of the cerebellopontile angle.

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[5] Allgemeine Neurologie, Part 2, p. 741 et seq.

Extracted from the American Journal of the Medical Sciences,
May, 1912, No. 5, vol. cxliii, p. 677.

TYPHOIDAL HEMIPLEGIA WITH REPORT OF THREE CLINICAL CASES AND ONE WITH NECROPSY.

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Hemiplegia following or occurring during typhoid fever is a comparatively rare complication, and any cases of this character are well worth reporting. It has been mentioned as early as 1860 by Gubler,[1] but since then, to my knowledge, there have been few cases published.

Among the more recent literature on this subject the paper of Smithies[2] takes up and classifies the symptoms, prevalence, age, time of occurrence, etc., in considerable detail.

In compiling the cases already published, with those described in this paper, several points are of interest.

The male sex was more often affected (70 per cent.) than the female (30 per cent.) in the 40 cases in which the sex was defined.

Where the side involved was tabulated, left hemiplegia occurred in 19 (44 per cent.) and right hemiplegia in 24 (56 per cent.).

In 28 cases of right side involvement aphasia was noticed in 23 (82 per cent.), while it is mentioned in only 3 (18 per cent.) of the 17 left hemiplegics.

The complication occurred before the age of ten years in 9 instances (24 per cent.), before twenty in 10 cases (26 per cent.), and in 19 (50 per cent.) before the age of thirty years.

Of those cases in which the time of onset was given, 10 (24 per cent.) occurred in the first and second week, 8 (20 per cent.) during the third week, 4 (10 per cent.) during the fourth, 15 (40 per cent.) during convalescence, and 2 (5 per cent.) so late after the attack as to render the dependence of the condition upon typhoid rather doubtful.

Athetosis was reported by Curschmann,[3] Osler,[4] and Barrett,[5] and is also present in my first case.

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[1] Arch. Gén. d. Méd., 1860.

[2] Jour. Amer. Med. Assoc., August 3, 1907, vol. xlix, No. 5. In this paper a very complete list of literature up to 1907 is given.

[3] Nothnagel's Handbuch, Band iii, 1 Teil.

[4] Johns Hopkins Hosp. Rep., 1899 to 1900.

[5] Report of Michigan State Asylum for Insane.

Visual defects have not occurred, with the exception of a left-sided amaurosis and nerve atrophy in an interesting case described by Benedikt.[6] Here there was also stuttering with the left-sided hemiplegia. Stuttering was also one of the symptoms of my first case. In Pigott's[7] case the pupils were dilated, the right larger than the left and less reactive to light than the left, also deviation of eyes to left. This was a left hemiplegia. A case of left hemiplegia reported by Parisot,[8] which ran a course of a few days, showed inequality of the pupils and strabismus. In Case IV there was deviation of the eyes toward the right with left-sided hemiplegia.

Sensory changes in most instances were absent or so slight as to be doubtful in value. Such changes were present in the case of Raschofsky's[9] and Case I of this paper.

An interesting case, more of a meningitic type, is described by Laure[10] in which, in addition to the typical signs of a meningitis, there occurred an aphasia with loss of ability to write, but no paralysis.

The hemiplegia occurring in typhoid does not differ materially from one following any other cause, and should therefore be readily diagnosticated. In some cases the onset may be acute, with convulsions or delirium or a rapidly developing coma or general restlessness with increase in the temperature, while in others it may be very gradual, extending over a period of hours or days or even weeks (Nothnagel), and in some instances reported there was no positiveness concerning the exact time of the occurrence of the paralysis.

This variety of onset is dependent to some extent upon the reactive or resistant condition of the patients' nervous system, but much more upon the character of the pathological lesion causing the condition. Thus, in the early cases where there is perhaps more likely to be a hemorrhage or an embolus, the onset is acute and there may be convulsions and coma. When, however, the symptoms are the result of a toxemia, there would more probably be a delirious condition of some duration, with temperature elevation in cases of an encephalitic character. In the convalescent stage there would be, more likely, a gradual onset, such as might occur in thrombosis, this lesion being in all probability the one which most frequently occurs during this stage of the disease.

A higher percentage of cases has been reported as occurring during the stage of convalescence, and this is due in all probability to the generally weakened condition of the patient at this period, the sluggishness of the circulation, and the condition of the vessels and the blood, tending to aid in the formation of thrombi in the cerebral vessels just as they do in the better known thrombotic processes elsewhere in the body.

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[6] Elektrotherapie, 1868.

[7] Maryland Med. Jour., 1909, lii.

[8] Rev. méd. de l'Est Nancy, 1909, xli.

[9] Wien. med. Woch. 1911, No. 39

[10] Rev. Neur., Paris, 1908, xvi.

The prevalence of this complication in the male sex is dependent upon a greater percentage of typhoid occurring in males, and also upon the fact that the male, through excessive venery and abuse of alcohol and tobacco, is more subject to such vascular changes as predispose to a thrombotic process.

The question of athetosis and choreic movements present in Case I, also reported by Curschmann, Osler, and Barrett, is of considerable interest as it is caused by involvement of some vessel other than the one so frequently affected. It might be due to an affection of the meningeal vessels, causing irritation, or of the vessel or vessels supplying the lenticular and thalamic regions.

The pathological condition most frequently occurring in these hemiplegics is in all probability a thrombosis, this being due in great part to the causes mentioned earlier in my paper. Thrombi were found in the Sylvian fissure branch of the middle cerebral artery by Dankin,[11] Hawkins,[12] Osler,[13] Barrett,[14] and in my fourth case. Embolic softening would in many cases be difficult to distinguish from thrombosis—cases of this nature were published by Curschmann[15] and Hruska.[16]

Parisot[17] reported a case in which the internal carotid was thrombosed at its entrance to the skull, and as a result the central part of the right hemisphere, the external capsule, and basal ganglion were totally destroyed, leaving only a shell of the cortex intact. Curschmann's case had a diffuse hemorrhage in the lenticular area.

Some of the hemiplegias, particularly the more transient and rapidly healing ones, may be caused by a toxemia affecting the cerebral cells, and this interpretation would more readily explain the great percentage of cases having a complete or almost complete recovery. Barié and Lian[18] referred to the probability of such an origin in their case.

Encephalitis may also be the basis of some of the cases, as in the one reported by Raschofsky.[19] Here the increase of the temperature at the time of the onset of the symptoms with restlessness and the recedence of the fever with the return to normal, led the author to consider an encephalitis as the cause.

The prognosis is dependent to a great degree upon the character of the lesion present. In those cases of toxemic or encephalitic origin the recovery may be almost complete. Where a hemorrhage or thrombosis is the basis of the condition, the change would be much slower and less complete. As a rule, the leg regains its power and function much earlier than the arm and hand. Aphasia, if present, may improve, but very seldom clears up entirely (Smithies). Treatment is largely symptomatic with the necessity of avoiding the use of any drugs or measures that might lead to hemorrhage. The late stages require the usual massage with general tonic treatments.

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[11] Lancet, April 23, 1892.

[12] Clin. Soc. Trans., 1889.

[13] Loc. cit.

[14] Loc. cit.

[15] Loc. cit.

[16] Präg. med. Woch., 1901, Nos. 41 and 42.

[17] Loc. cit.

[18] Bull. et. Mem. de la Soc. d. Hôp. de Paris, 1907.

[19] Loc. cit.

Papers have been written on cerebral and meningeal complications of typhoid by Du Castel,[20] Cabanes,[21] Barthelemy et Durbin,[22] Starkiewicz,[23] Diller,[24] Antony and Dofter,[25] and Laignell-Levastine.[26] Cases have been reported by Lesieur,[27] and Tondi,[28] also an atypical case of post-typhoid thrombosis by Pasteur.[29] I regret that I have been unable to obtain the records of any of these.

I am greatly indebted to Dr. William G. Spiller for the privilege of reporting the following cases, 2 of which were examined by me at the University and Philadelphia Hospitals, and a microscopical examination made in the third.

Case I.—E. K., aged twenty-two years. Admitted to the Philadelphia Hospital December 2, 1908.

Family history negative as regards possible affections of the nervous system.

Patient was well up to the age of six, when he developed typhoid fever. During the attack of typhoid fever he was confined to bed for three weeks. He remembers that he was widlly delirious during this illness, and wanted to jump out of the window. He also claims to have been unconscious during part of his illness, but does not know the duration of the unconsciousness. About two months after onset, and a week or so after beginning to improve, upon attempting to get up one morning he found that he could not use his left leg and arm, but does not know whether or not he could move his arm and leg while in bed. He was not able to walk for some time. His speech was affected so that people said he talked "tongue-tied," and he had trouble in swallowing. There were at no time any other symptoms of head involvement such as headache, vomiting, etc.

The athetoid movements of toes and fingers were present as long as he can remember, but he does not think they anteceded the attack. His left foot felt heavier and colder than the right, and he dragged it in walking.

Examination upon admission. Left arm was flexed and adducted, and was continuously and rhythmically flexed and extended. Fingers were flexed and athetoid movements were constant. The biceps was markedly contracted and prominent, and various muscles of the forearm could be seen contracting rhythmically. The left arm with a great deal of exertion could be raised to the level of the shoulder. The forearm could be flexed and extended slightly, and with very little strength. Pronation and supination were not possible. The handgrasp was very weak, as were the individual finger movements. The entire upper extremity was very spastic and resistant to passive movements; no atrophy. The left lower extremity showed retained power of flexing the leg on the abdomen, but this was greatly weaker than on the right. The leg could be flexed and extended at the knee, but no movement was possible with the toes, nor could the foot be elevated. The left foot was held in a partially flexed and adducted position, and continuous athetoid movements of the toes were present. Reflexes were markedly increased with ankle clonus and a Babinski strongly positive. Spasticity with resistance to passive movement was excessive. The leg was dragged in a spastic manner, but not swung in a circle, as it so often is in hemiplegics. Hypalgesia and hypesthesia were found over the entire left side, but no change in the temperature and position senses. Some slight asteriognosis was present in the left hand. There was no involvement of rectum and bladder.

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[20] Gaz. des Hôp. de Paris, 1910, lxxxiii.

[21] Bull. med. del.Algeriei, 1908, xix

[22] Gaz. med. de Nantes, 1906, xxiv.

[23] Gaz. lek Warszawa, 1906, xxiv.

[24] Jour. Amer. Med. Assoc., May 27, 1905.

[25] Arch. d. méd. et pharm. Paris, 1905, xlvi.

[26] Bull. et mem. des Hôp. de Paris, 1907, xxiv, No. 30.

[27] Bull. de.la Soc. méd. des Hôp. de Lyons, 1907, vi.

[28] Napoli, 1909, xxiv.

[29] Arch. Middlesex Hospital, London, 1909, xvi.

Examination March, 1910. There remains distinct foot drop and atrophy of tibialis anticus and the peroneal group. The calf muscles are contracted. The thigh muscles are still somewhat weaker than the right. At present the athetoid movements occur in the fingers and in the arm when the patient attempts to resist passive movements. There is some slight impairment of the lower facial movements.

Case II.—W. P. (No. 4218). Aged sixteen years. Male. Admitted to the University of Pennsylvania Dispensary December 11, 1905.

The patient was perfectly well up to four years ago, at which time he had a severe attack of typhoid. During this illness he had a left-sided hemiplegia, this having occurred while he was still in bed and running a temperature. The leg and arm were both affected; he could not move his fingers properly, nor the left side of the face. The patient said he could not talk for two days, although this might have been due solely to the involvement of the tongue and face. The eyes and ears seemed to be all right.

Examination on admission showed the left arm somewhat contractured and atrophied, with the reflexes increased, and the hands cyanotic and cold. No sensory changes were present. The power in the shoulder muscles was fair, but impaired. The patient had a distinctly spastic hemiplegic gait. All reflexes were increased with positive Babinski and some atrophy of the entire leg. There were no sensory changes.

Case III.—Mrs. L. G., aged thirty-two years, white. Admitted to the University Dispensary September, 1910.

The patient always had good health until four years ago (1906), when she had typhoid fever, and was in bed twenty-one weeks. During her illness her right arm and right leg would frequently drop over the side of the bed and would have to be lifted back. They were manipulated by the nurse, and when handled were always stiff and sore. When the patient got out of bed she experienced no especial difficulty in walking or standing, although she had to use a cane. The arm and hand were used only with great difficulty, and their original strength has never returned.

The right upper limb has never recovered in strength or size. She uses it awkwardly, and is forced to make more fine movements with the left hand. When she walks the right foot is dragged along the ground.

Examination. At rest the right side of the face shows some drooping in the lower half. Actively this is less marked, but still present. She winks the right eye less than left; wrinkles brow well. The tongue deviates to the right on protrusion. Ocular movements are good; reflexes normal. The upper extremities show marked weakness in all movements of the right arm, with all reflexes considerably increased. The entire limb is smaller than the left. There are fibrillary twitchings, but no sensory anomalies. The left arm is normal. There is no vesical or rectal disorder.

Lower extremities. Gait is unilaterally spastic. The patient drags the right foot. Individual movements are fairly well performed with right leg, very well with left. The whole right lower limb is distinctly smaller than the left. There are no fibrillary twitchings or sensory changes.

Reflexes. On the right the patellar tendon and Achilles tendon reflexes are greatly exaggerated. Ankle clonus is not demonstrable, probably on account of spastic contracture of Achilles tendon. The Babinski sign is present. On the left the reflexes are somewhat increased but not abnormal, and no Babinski.

Other than a slight thickness of speech during the first period of the attack, there was no disturbance of speech.

Case IV.—B. C., male, colored, aged twenty-four years. Admitted to Dr. B. F. Stahl's ward in the Philadelphia General Hospital (and examined by Dr. Spiller), January 6, 1906. Died January 10, 1906.

During the course of typhoid fever the patient sat up in bed and fell out toward the right, striking first upon his buttocks. He was put back to bed and Dr. Spiller, upon examining him, found a left-sided hemiplegia with deviation of the eyes away from the paralyzed side. The fall was regarded as due to cerebral hemorrhage or a thrombosis of the middle cerebral artery. The patient died five days later. Necropsy showed congestion of liver, spleen, and lungs, and typhoid ulceration of the ileum; apparently typhoid in the third week.

At necropsy a thrombus was found in the artery of the right Sylvian fissure and microscopically the areas supplied by this artery were found to have undergone softening with the presence of a great many fatty bodies or cells and cellular debris.

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BILATERAL SUPRANUCLEAR PALSY OF THE UPPER FACIAL DISTRIBUTION

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The present study includes two cases, one shown by Dr. Spiller to the Philadelphia Neurological Society in 1906,[1] and the other reported by Dr. Dercum[2] in a paper upon meningo-myelitis. The first of these merely presents a clinical record; the other supplied pathological material, and serial sections have been made by me of the medulla oblongata, pons, and part of the mesencephalon. Sections were also taken from most of the cranial nerves and the cord. The interesting feature that the cases present in common is the occurrence of a double almost complete facial palsy of supranuclear origin. In a survey of the literature of the past ten years, almost nothing could be found touching upon this subject. A double complete facial palsy as the result of upper segment disease is evidently, therefore, so rare that the pathological proof of this condition is a valuable contribution to the list of disease states of the central nervous system. Oppenheim, although he mentions the involvement of the upper branches of the facials in asthenic bulbar palsy, does not state that the parts supplied by these branches are ever affected in pseudo-bulbar palsy. These cases have also some value in the light which they throw on the nuclear origin of the upper facial branch supplying the orbicularis palpebrarum, corrugator supercillii and occipito-frontalis muscles.

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[1] JOURNAL OF NERVOUS AND MENTAL DISEASE, 1906, p. 594.

[2] *Idem*, 1905, p. 1.

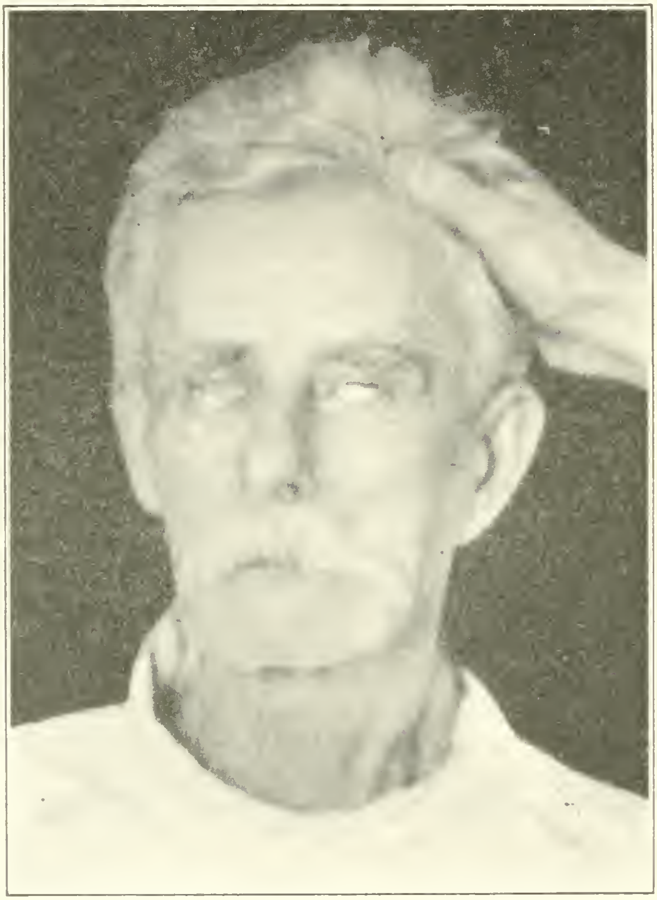

FIG. 1. Photograph of patient (Case 1), showing bilateral facial palsy including the upper distribution.

Mendell, Tooth, Turner and others believed that the fibres governing these muscles arise in the caudal portion of the oculomotor nucleus, and, after passing down the posterior longitudinal bundle, enter the facial nerve, most probably as it curves back of the sixth nucleus in close proximity to this bundle, to be finally distributed through its upper branch to these muscles. The weight of evidence has, however, more recently been thrown on the other side of the controversy as to the origin of these fibres, namely that they proceed from the facial nucleus itself. Bruce in an interesting and very impressive study of the facial nucleus, reviews the evidence supporting each point of view, and then adds recently observed facts in favor of these fibres arising from the seventh nucleus itself. He categorically states that this is the true solution of the controversy. The second case here presented confirms such a conclusion. It will be necessary to first give an account of the clinical aspect of these two cases.

The following is a description of the first case, which was presented clinically by Dr. Spiller to show the possibility of marked paralysis of the upper branches of the facial nerves, as well as of the lower, occurring in facial diplegia when lesions implicate bilaterally the cerebro-pontile tracts of the facial system, without involvement of the facial nuclei.

A man of 62 years, who denied venereal infection, was admitted to the Philadelphia General Hospital on December 6, 1905, presenting a left-sided hemiplegia, with some loss of power on the right side, and a bilateral facial palsy. The history that he gave was, that two months previously, while in good health, he first complained of pain around his heart and in both temporal regions, but continued to work. A week later he was compelled to go to bed on account of weakness in his arms and legs. Three days later, while making an attempt to get out of bed, he had a fainting spell, during which his wife noticed a twitching of the right side of the mouth. He was unable to reply for about five minutes, although he seemed to know that his wife was speaking to him. The next day it was first noticed that his face was drawn slightly to the right. The weakness, which had persisted in his arms and legs, gradually lessened, so that in about three weeks he was able to get up and walk about. He did not at this time notice any difference in the two sides of his body, and thinks he could close both eyes. Four weeks after the first attack, he had, while lying down after a heavy meal, a stroke which affected his left side. The arm was completely paralyzed, but the leg was not. The following day he was unable to move arm or leg, and noticed for the first time that he could not close either eye. The notes bearing on his nervous condition after admission to the hospital are quoted literally:

"Condition on Admission, 12–6–1905.

"Pupils are unequal. The left is slightly the larger. Both react to light, and in accommodation and convergence. All ocular movements seem to be normal, excepting convergence, in which the right eye is not carried in quite so far as is the left. There is a marked asymmetry of face, for the mouth is drawn very much towards the right. The left naso-labial fold is almost obliterated."

Notes were made by Dr. McConnell.

"The movement of the occipito-frontalis on the left is lost and much impaired on the right. Corrugators are similarly involved. The eyes can not be closed, but the right can be more nearly closed than the left. The left angle of the mouth is depressed and can not be elevated; the right is depressed but can be elevated slightly. When the mouth is opened the jaw goes distinctly to one side. The tongue is protruded to the left and is the seat of a fine fibrillary tremor. There is very slight or no difference in the movement of the temporal or masseter muscles.

"*Upper Limbs.*—The right arm is somewhat generally wasted. It is freely movable but the power is diminished. The grip is only fair. The right hand is the seat of a fine tremor, which is not increased by voluntary effort.

"The left arm is completely paralyzed, and is slightly spastic. The spasticity shows itself when the arm is fully extended. There is evidence of commencing contracture of the biceps. The proximal end of the upper extremity shows greater spasticity than does the distal end. The hand and fingers are completely flaccid. The reflexes are somewhat exaggerated on both sides. Movement is well preserved in right lower extremity. Spasticity of the left lower limb is slight and is much the same as that described in the left upper extremity. Both knee jerks are increased, but both Achilles jerks seem to be lost.

"Babinski's sign is present on the left; a normal plantar reflex is found on the right side.

"Sensation to touch, pain and temperature seems to be everywhere preserved.

"1–1–06. Patient closes right eye much better than he did.

Dr. Spiller made the following notes:

1–1–06. "Faradic irritation of the left seventh nerve just behind the ear produces distinct and prompt contraction of the whole distribution of left seventh nerve. Contraction is prompt to faradic current over all muscles on the left side of the face, contractions are about equally prompt on each side of the face.

"1–22–06. Patient can move the occipito-frontalis muscle on the right side much better and also closes both eyes much better. The right side of the mouth can be almost completely elevated.

"3–21–06. The patient closes his eyelids well on each side, and the resistance, when an attempt is made to raise the upper lid, is normal. He also wrinkles the forehead slightly on each side. Very little voluntary power is preserved in the lower distribution of the facial nerve on either side, but there is a little more on the right than on the left side. The left upper limb is completely paralyzed. Biceps and triceps tendon reflexes are exaggerated on the left side and very prompt on the right.

"The left lower limb is completely paralyzed except at the hip.

The patellar reflex is exaggerated on the left side, and is about normal on the right side. Ankle clonus is obtained on the left side, not on the right. The Babinski reflex is typical on the left side, but is not obtained on the right side. Left lower limb is quite edematous. Sensation for touch and pain is normal in all parts of the body.

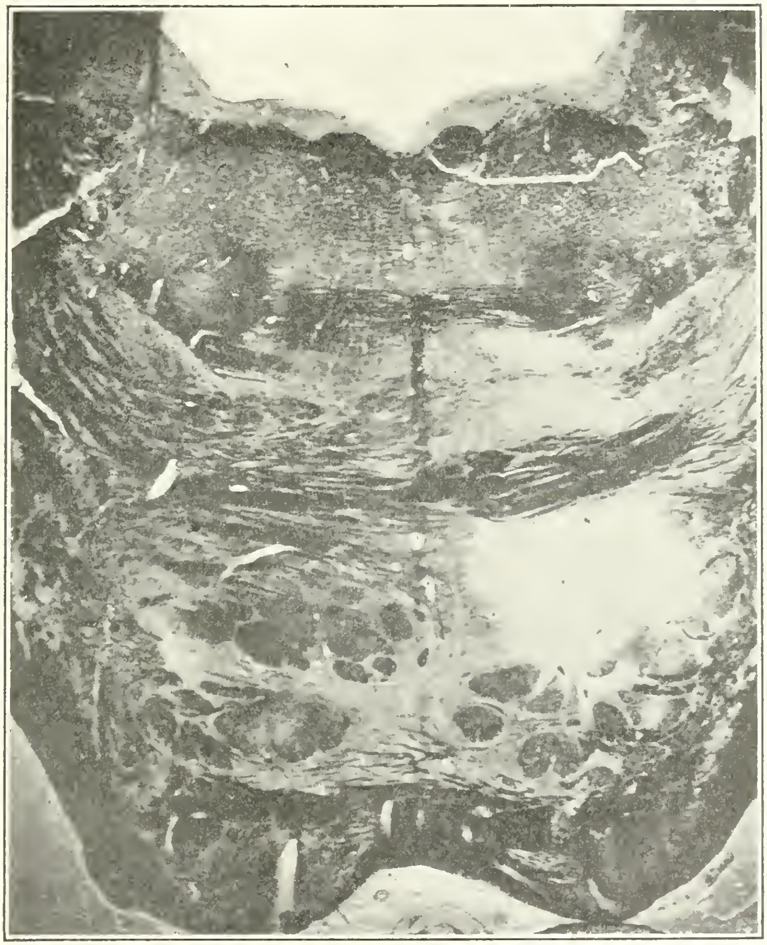

FIG. 2. Photograph of a section through the pons (Case 2) above the facial nuclei. Photograph by Dr. A. J. Smith.

"3–22–06. Voluntary power in both upper and lower right limbs is very fair."

There can be no doubt, of course, that there was a cerebral lesion on the right side, since the complete left-sided hemiplegia, with spasticity, is so typical (including the left side of the face). But the one factor that especially differentiates the condition of this side of the body from the usual hemiplegia due to a right-sided cerebral lesion is the inability to close the left eye and to raise and contract the brow. Mirallié, who has discussed the condition of upper facial palsy in cerebral hemiplegia, thinks that contrary to the ordinary opinion, the orbicularis palpebrarum, corrugator supercilii and occipito-frontalis are always slightly involved, and that this can usually be demonstrated by a difference in the size of the palpebral fissures, by the fact that the external end of the eyebrow can be felt nearer to the external inferior angle of the orbital cavity, by commanding the patient to raise and lower the brow (when, after several trials, it may be seen that it moves unevenly, possibly more slowly, and neither so far up nor down as does that on the sound side), and by having him test as to whether he can close the eye of the affected side alone when it is known that he could previously do so. A slight implication of the upper branch of the facial nerve unquestionably occurs in many recent cases of hemiplegia.

The apparent escape of these muscles supplied by the upper branch of the facial is due to the fact, as commonly understood, that the two sides are innervated together and have no real independent action. In other words, the only reason that these muscles apparently act normally in a cerebral hemiplegia is that the other side of the brain is normal. In the case under discussion, not only are these muscles opposite to the right-sided lesion palsied to such a marked extent that apparently the usual factor in making their movement possible (a normal condition of one side of the brain) is no longer working as ordinarily, but the other (that is the right) side of the face is also palsied. It was stated, also, in the above notes that the jaw deviated to one side when the mouth was opened—a phenomenon that does not usually occur in an ordinary hemiplegia, although weakness of the muscles of mastication is sometimes detected. We may assume that the lesion on the right side of the brain was above the fifth nerve nucleus, since the pterygoids and all the muscles supplied by the facial nerve were affected, as well as the whole muscular system on the left side below these. As said before, a lesion on the right side alone, above the fifth nucleus, would not, however, cause such a marked paralysis of the muscles supplied by the upper facial and the fifth nerve on the left side as occurred, and we are, therefore, driven to inquire whether there is not also a lesion on the left side of the brain, which would make impossible the ordinary synergetic action of these muscles on the left with their homologues on the right, when the paths leading to the innervation of the latter are intact. And this inquiry is seemingly answered in the positive by the fact that there was a facial palsy on the right —less to be sure than on the left, but still present to a marked degree and including, at one time in the history of the case, the upper portion of the face. There was some weakness of the extremities on the right, but not a complete paralysis. The indications are, therefore, that the left sided lesion was much less extensive than that on the right side, and that it was also above the facial nucleus. This position, that the lesions were above the seventh and even the fifth nuclei, is, of course, made much more secure by the fact that both facials responded normally to the faradic current and that nowhere was there any disturbance of sensation. We may also assume it to be most likely that at least one of these lesions was below the third nucleus, since the nerve there arising remained unaffected on both sides, and a double lesion above that area would in all probability work the same results in the muscles supplied therefrom as are produced by a double lesion above the cooperating facial nuclei in the region innervated by them, although the fibers passing to the third nucleus might escape.

The case to follow, with the pathological study, will show whether the explanation of the symptoms in this, by the assumption of lesions in the pyramidal tracts on both sides above the facial nuclei, is justified. It is also a case of syphilis as the history of the one just studied clearly shows it to be.

The patient, a German of 38 years, gave a history of having had, four months before admission to the Philadelphia General Hospital, a sudden attack of dizziness, with dimness of vision, but no loss of consciousness; and weakness, but not paralysis of the right leg. He improved very much, but a few days later he had another attack of dizziness, which was followed by loss of speech, inability to swallow, marked weakness in the left arm, and, to a less extent, in the left leg. He stated that his mouth was drawn to the left. After three days his speech returned, but the loss of power in his left extremities persisted until his admission on June 6th, 1902. He then could move his right arm and leg in all directions with considerable strength, but there was loss of power on the left side, which was greater in the arm. This was accompanied by increased tendon reflexes, even clonus in the lower extremity, and some wasting of the muscles of the arm, shoulder and thigh. This wasting was also especially noticed in the interosseous muscles of the hand. The arm showed contractures—the forearm was flexed on the arm, and the fingers on the hand. The tendon reflexes were increased on the right, and there was also ankle and patellar clonus on this side. There was a Babinski reflex on both sides. There were no eye symptoms. The tongue deviated somewhat to the right when protruded. The mouth was drawn slightly to the left but on voluntary effort it was moved better to the right. No difference, on the other hand, was noticed when the man smiled. The pterygoids and masseters were apparently weakened. He was unable to completely close either eye; forcible effort caused rapid tremors of both eyelids. At first no paralysis was noted in the occipito-frontalis or corrugator supercillii, but later the action of the left occipito-frontalis became evidently diminished, and the brow on that side was relatively smooth. When the note of this last observation was made it is stated that there was an appreciable change in the symptoms—that his right arm and leg became distinctly ataxic, and that the left leg was so spastic and muscular regidity so great that movement was decidedly restricted. There is then a note that the "left eyeball occasionally drops outward as though from weakness of the left internal rectus," although the statement is elsewhere repeatedly made throughout the notes, that the extraocular muscles were normal. The tactile, thermal, and pain senses are always stated to have been normal, as was also the sense of position. The abdominal and cremasteric reflexes were more active on the right side, and there was a jaw jerk present. There was diminished faradic contractility in the interosseous muscles and abductor policis of the left hand. The right leg, as well as being atactic and somewhat rigid, had also become somewhat weaker. On October 17 the man had another attack of dizziness which caused him to fall. After this his speech, which had been slightly thick before, was a little worse.

The examination of the eye grounds by Dr. Crosky in July revealed contracted arteries (dilated veins and slight hyperemia of the left disc, but no signs of neuritis, atrophy or extravasation. Dr. de Schweinitz's examination of the eyes on October 29th is as follows: "Both discs now distinctly degenerated, giving the appearance of a low grade interstitial neuritis. The veins are very full and the arteries are contracted. The reaction of pupils to light is normal. There is no diplopia. Rotation of eyeballs is normal. There is partial paralysis of each orbicularis palpebrarum. The patient is unable to completely close the eyes."

On November 22 Dr. Spiller dictated the following notes: "It is noted since the last examination (about a month ago) that the condition has become much worse. The voice is weaker and more indistinct. The patient is now unable to remain out of bed. When sitting in a chair he falls over. He makes no complaints. His face has a mask-like appearance. He is unable to close the eyelids completely, and when he makes an effort to do so, the palpebral fissure on each side remains open for from one eighth to one quarter of an inch. He has much difficulty in showing his teeth, and does it very imperfectly. He cannot draw up the corner of his mouth on either side. His tongue deviates well to the right when it is protruded, and is unusually thin, but equally so on both sides, and shows no fibrillary tremor. His pupils are equal, possibly the left is a little larger than the right. His irides react promptly to light. The movement of the eyeballs is normal in all directions. There is no nystagmus. Every now and then, during the examination, there are contractions of the facial muscles, such as occur in laughter, and this takes place without any-

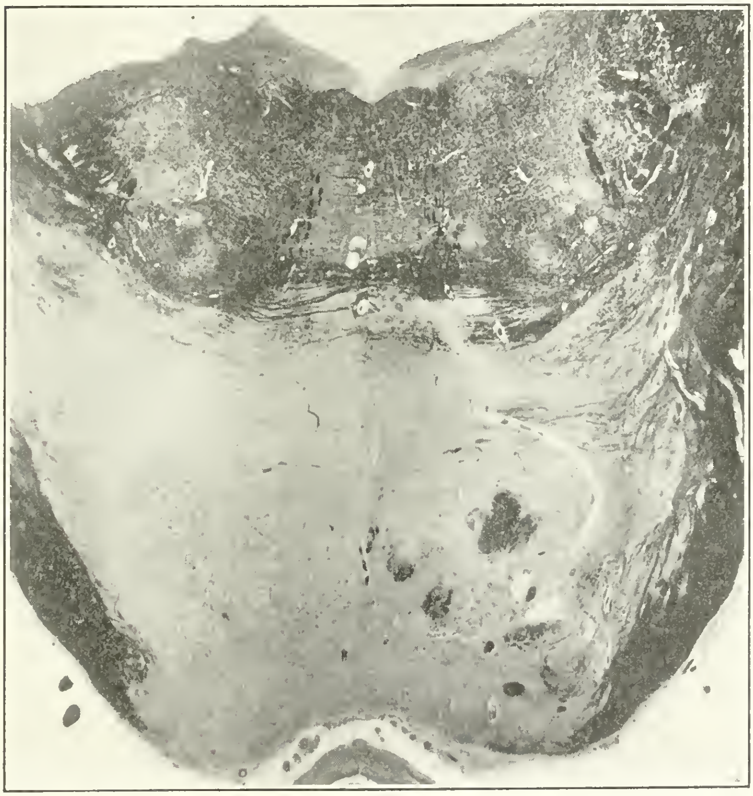

Fig. 3. Photograph of section at a lower level than that represented in figure 2. Photograph by Dr. A. J. Smith.

thing to amuse him. He can move the right upper limb freely, but the movement is ataxic, and he has difficulty in placing the first finger of his right hand on the end of his nose. The left upper limb is paralyzed, and he can move it very slightly at the elbow. The contractures at the left elbow and in the fingers are very marked. The biceps and triceps tendon and wrist reflexes, in the left upper limb, are much exaggerated. These reflexes in the right upper limb are slightly exaggerated.

Sensation for pain is preserved in both upper limbs over the trunk and in the face. Sensation for touch can not be accurately determined on account of his feeble mental condition. He has incontinence of urine and feces. The left lower limb is paralyzed, there is contraction in extension and it is impossible to flex the left leg passively at the knee. The patellar reflex on the left is exaggerated but no movement of leg can be obtained because of the contractures. The patellar reflex on the right is much exaggerated. Ankle clonus is distinctly present on both sides. Babinski's reflex is very marked on both sides—the big toe is moved promptly upward. Sensation for pain is preserved in both lower limbs."

Further notes while he was in the service of Dr. Dercum were on the following dates.

December 26, 1902: "He is unable to raise himself in bed. He can, however, turn over on the left side by grasping the rail of the bed with the right hand. He says that he is comfortable and has no pain. He closes his eyes more incompletely than before; the palpebral fissure on the right side now remains open for about one third of an inch, on the left for about one quarter of an inch. He has less power to show his teeth than before, and both angles of the mouth can be but feebly retracted, the left to a less extent than the right. When he laughs, the angles of his mouth are retracted much better and equally. The tongue is protruded only slightly and with difficulty, and it deviates markedly to the right. There is no change in the pupillary phenomena or ocular movements. Patient moves his right arm in various directions but the movements are ataxic and jerky and his grip is very weak. He cannot move the left upper extremity, except to make a faint movement of flexion at the elbow. There is no motion of the hand or fingers. Contractures of shoulder, elbow, wrist and fingers are present as at previous examinations. The reflexes, sensation and sphincters are as before. He is able to move his right leg feebly in various directions, and also the foot and toes. He can move the left leg feebly, flexing it slightly at the knee. The left leg can now be moved passively at the knee, as it is not quite as rigid as at the last examination."

On January 22, 1903, he had a rise of temperature, and on the following day, after showing general failure, which was accompanied by a small and very rapid pulse, he died. His respirations had remained normal in frequency, his lungs clear, and his abdomen had shown no distention.

The pathological study includes serial sections of a portion of the mesencephalon (two sets of about ninety sections each in the region of the third and fourth nuclei), and of the lower half of the pons and the upper portion of the medulla oblongata (to the number of about three hundred and sixty sections), also sections of the third, fourth, sixth, seventh and eighth cranial nerves, and of the cervical, dorsal and lumbar portions of the cord. It should be mentioned that owing to the material having been kept a long time in Müller's fluid, the writer was compelled to rely for cell study upon the acid fuchsin stain. The tissue throughout shows intense round cell infiltration. The vessels, frequently engorged, are thickened and surrounded by a still more intense grade of round cell infiltration. Some of these vessels thus appear to be the starting point of softening. The pia-arachnoid is thickened and presents intense infiltration by these small round cells. Immediately beneath this, the neuroglia seems to be somewhat proliferated and presents a dense appearance over the pyramids. The vessels at the base of the brain also show marked thickening. There is some proliferation of ependymal cells. These same general changes occurred in the cortex, although they were there less pronounced than elsewhere.

Region of third nucleus in mesencephalon.

There is a degeneration of the extreme inner portion of the right crus. Through the region of the mesencephalon there are numerous small areas of beginning or complete softening. In the upper sections there are, on both sides, small areas of infiltration, in some sections reaching a real softening, between the posterior longitudinal bundles and the red nuclei; that is, ventral to the former and dorsal to the latter. On the left, a small area of softening in the red nucleus itself connects with the patches impinging upon and to a certain extent infiltrating into the posterior longitudinal bundle. On the right, there is considerable destruction of the posterior longitudinal bundle itself. On the left, in a few of the sections, the patches of softening encroach upon the latero-dorsal portion of the third nucleus, probably destroying a few cells, and there is also a patch along the median side of the red nucleus, just outside of and catching a few fibres of the emerging third root on that side. In addition to these, there is also, on the left, a collection of small patches in the outer half of the substantia nigra, extending to a very slight extent into the pyramidal tracts. There are in this series eight, or ten, or more different foci of degeneration, but several of these, in some sections, merge more or less into each other, so as to form larger areas. There is, then, a very slight destruction of the right third nucleus in the extreme lateral portion of its dorsal part, owing to a patch of softening which also involves some of the posterior longitudinal bundle, and a still slighter involvement of the corresponding portion of the left nucleus, with an additional patch of softening to its outer side and encroaching slightly upon its ventral portion. The cells otherwise throughout the nuclei have a normal appearance with the exception of containing an excessive amount of pigment. Only a few cells in the immediate vicinity of these softened patches are smaller and present eccentric nuclei. As others have observed, these third nuclear cells seem to have a high degree of resistance, for some of them appear normal even when surrounded by softening.

Region of fourth nucleus in mesencephalon.

There are areas of softening and degeneration here which are continuations of some of those above, as well as new foci. The patches of softening (there are three) in the left crus, are here more ventral, so that they are in the pyramidal tract at about the junction of the middle and outer thirds of the pes. Certainly the width of the area destroyed is one-sixth of the whole pes. Of course, the same degeneration of the inner third of the right crus here continues, and the inner portion of the right posterior longitudinal bundle shows degeneration throughout these sections. In addition to these lesions there is a patch of softening, to the right of the median line, in the fibres of the brachium, at the decussation, before the red nucleus has appeared. There is also a very small focus in the left fourth nucleus, destroying a few of its cells. In only one place are a very few of the cells found with somewhat excentric nuclei, otherwise they are normal in appearance throughout on both sides.

The upper level of the next series begins below the emergence of the fifth nerve roots, but well above the upper limit of either facial nucleus, and above the sixth nuclei and the roots passing therefrom through the pons. Fifty sections above the upper limit of the facial nucleus, there is, an area of softening in the posterior part of the right pyramidal tract destroying nearly half of this tract, a smaller area on the same side just ventral to the trapezoid fibres, and an area of degeneration in the formatio reticularis, just anterior to and to the outer side of the posterior longitudinal bundle. On the left the pyramidal fibres show a slight amount of degeneration, and there is a beginning small patch of softening. These areas increase in size as lower levels are reached, until, at the beginning of the facial nucleus, on the right (which in these sections is a little higher than on the left), practically the whole of the right pyramidal tract has been destroyed, only one band of partially degenerated fibers remaining. And on the left, at least two thirds or three fourths of the pyramidal tract has been destroyed or shows degeneration. There is a tendency at this level for these separate patches of softening to merge, so that the right and left join as well as the two areas on the right. All of the transverse fibers ventral to the trapezoid bundles are here destroyed, and the fillet on the right is much affected. There is also a small patch of softening just median to the sixth root (possibly catching some of its fibers) and just anterior to the posterior longitudinal bundle, which is very slightly encroached upon. A few sections lower, when the left facial nucleus is first seen, there is practically one large area of softening of nearly the whole ventral portion of the pons, leaving no pyramidal fibers on the right and very few on the left. The facial nuclei extend through this series for a hundred and thirty to forty sections. This extensive softening continues in the ventral portion of the pons as far as its lower border, which is nearly the lower limit of the facial nuclei, and throughout this area all the transverse fibers in the ventral part of the pons are interrupted. The pyramidal tracts in the upper part of the medulla oblongata show some medullated fibers on the left, but so few on the right that they could be counted. The actual softening does not extend below the pons, except for a very short distance in the most ventral transverse fibers on the right side.

The facial nucleus presents normal cells throughout on the right, but on the left while the nucleus as a whole appears to be in a normal condition, there is occasionally found a cell showing some chromatolysis or a slightly excentric nucleus or both of these conditions. This alteration is insignificant. But both seventh nuclei look pale in places, from evident degeneration of the terminal fibres of the upper segment.

The cells of the sixth nuclei also appear fairly normal, with the exception of a very few cells showing a tendency to excentric nuclei. It is possible, however, that the right nucleus has a scarcity of cells, and that they are smaller than those on the other side.

The sections of the cranial nerves were made below, but near their exit from the brain. Both eighth nerves appeared to be in a condition of very fair preservation. A careful study of both seventh nerves (just peripheral to their exit from the junction of the pons with the medulla oblongata) shows them to be normal. The right sixth is partially but distinctly degenerated; the left is also affected but to a lesser degree than the right. The fourth nerves present slight degeneration, more especially the left. There is marked degeneration in both third nerves.

The spinal cord presents the same general changes (except distinct areas of softening) that are seen in the medulla oblongata and pons, namely meningitis and infiltration and thickening of the vessel walls. There is moderate general peripheral infiltration, much less marked in the posterior portion of the cord. Throughout the cord there is very marked degeneration (with infiltration) of both crossed pyramidal tracts and of the right direct pyramidal tract, with slight degeneration of the left direct pyramidal tract.

We have, then pathological evidence in this second case of a condition that we have assumed in the first, namely, a double facial palsy due to lesions interrupting the pyramidal fibres before the facial nuclei are reached. The facial nuclei, as far as the cells constituting them are concerned, were seen to be practically normal throughout, with the exception of a very few cells on the left, showing slight degenerative changes, and nowhere do any of the areas of softening impinge upon either the facial nuclei or fibers; moreover both facial nerve trunks were normal. We have, therefore, pathological evidence that the bilateral facial palsy, including the distribution of the upper branch of both seventh nerves, was in this case due to lesions involving both cerebro-pontile segments, with an escape of the lower segment. And such a double lesion must evidently produce a marked palsy of the upper face, since the factor that prevents the appearance of a unilateral upper face palsy in ordinary hemiplegia is eliminated, namely the healthy condition of the upper segment of the motor system on one side innervating these muscles through synergic action. But even in hemiplegia this synergic action preserves a normal condition in the upper facial muscles more apparent than real. Investigation according to Mirallié would as a rule reveal at least some loss of tone there. As previously mentioned, Bruce has surveyed the evidence as to the origin of these fibers traversing the facial nerve to the frontalis, corrugator, and orbicularis palpebrarum muscles, and concludes, with the support of his own case, that they come from the dorsal part of the facial nucleus and entirely from the same side as that to which they are distributed. Mendell, after section of the upper facial nerve in young rabbits and guinea pigs, found atrophied cells in the lower part of the oculo-motor nucleus of the same side, and traced their connection with the facial nerve root, as it winds back of the sixth nucleus, through the posterior longitudinal bundle. This view of the supply to the upper facial muscles seemed to satisfactorily explain the comparative escape of the muscles of the upper face, while those of the lower part of the face are involved, in bulbar palsy and amyotrophic lateral sclerosis. Bruce, quoting Willbrand and Saenger, presents a number of cases, with the authors' names, of ophthalmoplegia externa associated with involvement of the orbicularis palpebrarum or the other upper face muscles. But he remarks that these cases were only clinical reports and in the absence of post mortem demonstrations that there was alone degeneration of the third nuclei, cannot be held as valuable evidence that this nucleus gives rise to the fibers of the upper facial. He suggests that these associations may occur without a single lesion. However, even though it is accepted that the upper facial fibers take origin only from the facial nucleus, there is no denying a very close association between the cells giving origin to them and the third nucleus, and it is quite likely that there are fibers running through the posterior longitudinal bundle, from the latter to the former, which carry reflex impulses and also to a certain extent help to maintain the tone of the upper facial distribution.

Against Mendell's theory are a number of observers (Cassirer and Schiff, Bernhardt, Sauvineau, Siemerling and Boedeker, Parhon and Nadejde, Marineseo, Kotelewski, and Minea), who have either found no degenerated cells in the third nuclei in upper facial palsy; or who have found no upper facial palsy in degeneration of the third nuclei, even in its caudal part; or no axonal reaction in the third nucleus, either on section of the upper facial or the whole nerve, but a degeneration in some of the cells of the facial in the former or all in the latter case. Bruce's case demonstrates the same fact (as does that of Fowler and Painy), and he concludes rather positively, as before stated, that the upper facial fibers arise from the facial nucleus, in probably its dorsal part, and that there is no crossed origin for the facial. In the second case here studied it has been shown positively that the facial palsy, at least that of the lower face, was of cerebro-pontile segment origin, since the facial nerves and nuclei had escaped any destruction, since there were spasmodic contractures during life, and since there were sufficient lesions in the pyramidal tracts above the facial nuclei to explain the symptoms. If it were assumed that while the palsy of the lower part of the face was indisputably of cerebro-pontile segment origin, that of the upper part of the face was of lower segment origin (that is in the third nucleus or fibres arising therefrom) not only would that be a strained explanation, but one not consistent with the facts observed. It will be recalled that there was a degeneration of the extreme inner portion of the right crus and that it is not possible to say whether any of the pyramidal fibers to the third nucleus were caught or not, also that the softening in the outer portion of the left crus had not at this level penetrated the pyramidal tract, but was behind it. Therefore, even if any of the fibers in the pyramidal tract to the third nucleus were involved it was on the right side only, and the palsy on both sides could not be accounted for. But, following Obersteiner, it is extremely unlikely that the pyramidal fibers were affected even on the right side above the third nucleus. Moreover, there were detected no palsies at all in any of the muscles controlled by the third, fourth, or sixth nerves. It is evident, therefore, that there would be no warrant whatever for claiming the upper facial palsy to be supra-nuclear in origin if the fibers of the superior facial arise from the third nucleus. If this were the nucleus of origin, we would, therefore, be driven to the hypothesis that the complete facial palsy was caused by lesions of both pyramidal tracts at and above the level of the facial nuclei, with double lesions in either the third nuclei in its lower part or in the posterior longitudinal bundles. The study of the third nuclei do not at all warrant the conclusion that the palsy was caused by lesions there. There is somewhat more evidence in the destruction of portions of the posterior longitudinal bundles, but the limited degeneration seen throughout the mesencephalon in the right bundle is not seen throughout this area in the left one. In the pons the posterior longitudinal bundle looks quite normal on the left, but that on the right is possibly a shade lighter, although the difference is barely perceptible. It is, therefore, only on one side, the right, that an assertion could be made that there was degeneration in the posterior longitudinal bundle extending from the mesencephalon to the level of the facial nucleus, and that scarcely perceptible in this portion of the pons. But, even granting that it is sufficient on the right side to account for the upper facial palsy on that side (which it really does not seem to be), it still leaves us without a consistent reason for that on the left side.

Finally, this argument to show that the upper facial palsy was not due to disease in the lower segment of the facial motor system, either by destruction in the third nucleus or by interruption of the fibers supposedly travelling therefrom to join the root of the facial nerve as it winds back of the sixth nucleus, is rendered somewhat superfluous by the fact that both facial nerves were normal soon after their emergence from the ponto-medullary junction. We, therefore, find ourselves forced to accept the evident cause in the destruction of both pyramidal tracts just above the facial nuclei, and to positively assign the origin of the upper facial fibers to some part of the facial nucleus. Although the evidence as to this origin has been strongly presented by several observers, it was felt that this phase of the case under pathological study, is of value in helping to establish still more firmly a fact formerly much disputed.

The study of the sections in this case also shows another interesting though well known anatomical feature, and that is, the addition of new fibers to the pyramidal tracts as these travel downward. In the lower part of the pons there is absolute destruction of all pyramidal fibers by softening on the right side, and yet, as we study lower sections, we see a few fibers appearing in the degenerated anterior pyramids and these increase in number as we proceed lower in the medulla oblongata. This is apparent on both sides but it is more evident on the right side because the destruction was there absolute, which of course eliminates the question of judgment, as to the comparative number of fibers. This increase is also noticeable in the cord, in which both the direct and crossed pyramidal tracts show many more fibers than one would be led to expect from the destruction above. The pyramidal tracts do not differ from other great tracts, in the fact that they are constantly losing and even gaining some new fibers in their progress downward, and from a study of these sections it would appear that after they have reached the cord they gain proportionately fewer the farther down their course is followed.

The Treatment of Persistent Pain of Organic Origin in the Lower Part of the Body by Division of the Anterolateral Column of the Spinal Cord

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THE TREATMENT OF PERSISTENT PAIN OF ORGANIC ORIGIN IN THE LOWER PART OF THE BODY BY DIVISION OF THE ANTEROLATERAL COLUMN OF THE SPINAL CORD*

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REMARKS BY DR. SPILLER

Occasionally the pain from tumor of the cauda equina is so intense that complete division of the spinal cord has been suggested for its relief, and it is only recently that I have seen a case in which this measure had been proposed. If division of the anterolateral columns alone will remove pain, it may be possible, if the other parts of the cord be left intact, to avoid paralysis of the lower limbs and of the bowels and bladder by this operation. It probably would produce ataxia. Such an operation may be considered only in extreme cases when life has become almost a burden because of severe suffering.

A. Schüller[1] has suggested that as a substitution operation partial division of the spinal cord might be employed instead of section of the posterior roots for spasticity and gastric crises. The posterior columns, he suggests, might be cut for spasticity either alone or with the direct cerebellar tracts; the anterolateral columns might be cut for gastric crises. He reported no cases in which this operation was performed, and he advised it for these two conditions.

I would suggest that the operation of division of the anterolateral columns is applicable for pain independent of gastric crises, and that it may be much more than an *Ersatzoperation* for division of the posterior roots. So far as I know, the case reported in this paper is the only one in which this operation has been done.

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* Read before the College of Physicians of Philadelphia, April 3, 1912.

[1] Schüller: Wien. med. Wchnschr., 1910, p. 2292.

On what is this operation for the relief of persistent pain based? Are fibers of pain confined to one system within the spinal cord, and if so, can pain sensations be conveyed by other systems when the fibers usually employed for this purpose are destroyed? These are important questions.

Head and Holmes,[2] in their recent Croonian lectures, refer to the work of Rivers and Head, in which it was shown that beneath the skin, independent of all "touch" and "pain spots," lies an afferent system capable of a wide range of functions. Pressure which in ordinary life would be called a touch can be appreciated and localized with considerable accuracy. Increase of pressure, especially on bones and tendons, will cause pain. In the peripheral mechanism, therefore, there are two independent mechanisms for the initiation of pain. Within the spinal cord, however, the conditions are different. All impulses capable of generating pain become grouped together in the same path and can be disturbed simultaneously by an appropriate lesion of the spinal cord. In the same way, sensibility to heat or cold may be lost independently of one another, showing that all the impulses on which they are based have been sorted out into two functional groups, each of which passes by a separate system in the spinal cord. It should be possible, therefore, to divide the tracts for the conduction of pain sensation with little damage to the rest of the cord, except that the fibers of temperature sensation probably would be implicated. It is questionable whether objective disturbance of sensation would be produced by such an operation, and, if it were, whether it would be persistent.

Fabritius[3] recently has stated that little attention has been paid in neurology to the late results of interruption of the temperature and pain tracts in the spinal cord. It is in general assumed that the disturbance in these sensations gradually disappears, but if one attempts to determine on what this opinion depends he meets with difficulty. Fabritius was not able to find any extensive investigation of this subject, and he attempts to fill the gap by collecting cases from the literature. The summary of his findings is that after stab-wounds of the spinal cord no regularity exists in the restoration of pain and temperature sensations. In the majority of the cases (twenty-three out of thirty-five) temperature sensation seems to have been permanently lost. Pain sensation returned more frequently, but in diminished intensity, namely in eighteen out of thirty-eight cases, but was permanently lost in sixteen cases. It is questionable whether all the statements regarding sensation in these cases can be accepted as accurate.

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[2] Head and Holmes: Lancet, London, Jan. 6, 1912.

[3] Fabritius: Monatschr. f. Psychiat., u. Neurol., January, 1912

Rothmann,[4] in a paper read at the recent meeting of the Gesellschaft deutscher Nervenärzte, gave as the results of his investigations that pain sensations are conducted essentially through the anterior part of the lateral column, and that the gray matter may be concerned in this function, but concerning the latter statement the report is not very definite.

The work of Petrén[5] and my own studies[6] of the location of pain fibers within the spinal cord lead to the conclusion that fibers for the conduction of pain are located in the anterolateral columns.

In order to make a test of this reasoning in a very severe case of tumor of the lower part of the cord, when pain was intolerable, I referred a patient to Dr. Edward Martin for division of the anterolateral column on each side. Dr. W. B. Cadwalader and Dr. J. E. Sweet later performed the same operation on dogs to further our knowledge in respect to the function of these columns.

Patient.—A man, aged 47, was admitted to the Philadelphia General Hospital, March 8, 1909, in my service, and came later under the care of my colleagues on the staff. He had had pain for two months in the knees and ankles, and at the time of admission had pain about the pelvis. The pain increased, and by August, 1909, he complained greatly of it and had almost complete flaccid paralysis of the lower limbs, with loss of tendon-reflexes, and paralysis of bladder and rectum. He had anesthesia over the buttocks and external genitalia and down the back of the thighs. The lower limbs were atrophied and faradic contractility was diminished.

Diagnosis.—In November, 1909, introduction of the finger into the rectum caused no contraction of the internal or external sphincter. Dr. W. P. Hearn operated and found a growth on the left side and lower part of the spinal cord about as large as a goose-egg, the cord being implicated by the tumor. It was regarded as malignant and irremovable. In January, 1910, the man came again into my service and notes taken by me at that time record that he had very slight movement at each hip and in the left knee, and no movement elsewhere in the lower limbs. These limbs were greatly wasted. Sensations of touch, pain, heat and cold were lost in the back of each thigh and in all parts of the legs below the knees, although deep pin-prick seemed to be felt in the left calf and left foot. The tendon-reflexes were lost and the legs were contracted on the thighs and the thighs on the abdomen. Sensation in its various forms was preserved in the abdomen and front of the thighs. The man suffered greatly from pain in the lower limbs and required morphin every night for relief.

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[4] Rothmann: Monatschr. f. Psychiat. u. Neurol, January, 1912, p. 81.

[5] Petrén: Arch. f. Psychiat., xlvii, 495.

[6] Spiller: Univ. of Penn. Med. Bull., July and August, 1905.

Operation.—The division of each anterolateral column was performed by Dr. Martin, Jan. 19, 1911. On January 22, there was great relief of pain in both lower limbs. The patient appeared very grateful for the relief from suffering and received only 1/6 grain morphin on the day following the operation and a similar amount two days later, and this was given for the pain caused by the operation. The intern on the surgical service, who had the opportunity of observing the man constantly, believed that he was not suffering pain in the lower limbs. Sensation in its various forms objectively tested did not seem to be much impaired above the hip-joints. Pain was felt occasionally in the lower limbs during the three weeks following the operation, but the man was positive that it was less than before the operation.

Course.—In March, 1912, when he was again under my care, he lay in bed with his lower limbs greatly contracted and atrophied. Objective sensation was lost in the front of the thighs, but pain and pin-prick sensations were keen over the lower abdomen. His countenance expressed no pain and he never complained unless he was asked concerning his condition, when he would reply that he sometimes had pain. As he is a foreigner and not very intelligent, and his condition is pitiable, it is hard to judge whether he really has any pain from the tumor. The operation seems to have been successful in the diminution of pain. I should be unwilling to form an incorrect judgment regarding this case. When one remembers, however, that the pain from a malignant tumor of the lower part of the cord usually is excruciating and continues as the tumor implicates additional posterior roots, I think it may be said that the peaceful expression of this man's face is evidence that he has been greatly relieved by the operation. He has certainly not been made any worse, as his condition before the operation was very grave.

REMARKS BY DR. EDWARD MARTIN

The problem presented by Dr. Spiller was the making of a transverse cut into the spinal cord, roughly 2 mm. in length, of a similar depth and with its posterior end 3 mm. anterior to the entrance of the posterior root, this cut to be bilateral. The only special instrument required was a small thin-bladed cataract knife, double-edged on its angled point, the latter being about 5 mm. long. Since in this individual case there was no need for conservation of the bone, the laminæ and spinous processes of the sixth, seventh and eighth dorsal vertebræ were removed. The dura was split, retracted by threads, the cord was slightly lateralized by passing threads about the posterior roots and gently using them as tractors, and the cord incision was made on each side with very little bleeding. The wound was closed without drainage and, in so far as it was concerned, the after-course was uneventful.

The approach to the cord is greatly simplified and expedited — although it was not done in this case — by the use of the Hudson trephine and the Cryer bone-cutting instrument. By the help of these tools it is possible rapidly and safely to obtain, by unilateral resection, preserving the spinous processes, an exposure adequate for many forms of dural and cord intervention. It would certainly be so for a limited section, such as was practiced in this case, or for section of posterior roots. Such exposure leaves little or no subsequent crippling so far as the spinal column is concerned and is much simpler than an osteoplastic flap. In case of unilateral pain, uncontrollable by other means, cord section on the side opposite to that in which the pain is felt and sufficiently high to allow for the decussation of sensory fibers should be adequate.

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EXPERIMENTAL WORK ON THE FUNCTION OF THE ANTEROLATERAL COLUMN OF THE SPINAL CORD*

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The present investigation was undertaken at the suggestion of Dr. William G. Spiller, in order to determine what symptoms would be produced by destroying the anterolateral column, including Gowers' tract. In Gowers' original observation[1] he referred to a group of fibers situated in the anterolateral columns of the cord, which underwent degeneration from a lesion of the eleventh thoracic segment. He traced these degenerated fibers upward as far as the cervical region, and believed that they were concerned in the transmission of painful stimuli from the opposite side of the body.

Since that time innumerable clinical and pathologic studies seem to confirm Gowers' first impression. Experiments on animals, however, have not always lent support to this opinion, the results in some instances having been very confusing and even contradictory; indeed, Mott,[2] after cutting this tract in monkeys, concluded that its function was unknown. Bing,[3] experimenting on dogs, observed ataxia without disturbance of cutaneous sensation, but in his cases, as in Marburg's,[4] the area destroyed was chiefly the dorsal cerebellar tract or close to it. Bruce and Schäfer[5] conducted similar experiments on monkeys in which only the ventrolateral region was destroyed; they not only found sensation entirely normal.

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* Read before the College of Physicians of Philadelphia, April 3, 1912.

[1] Gowers: Diagnosis of the Diseases of the Cord, 1879.

[2] Mott: Brain, 1895, xviii, 1.

[3] Bing: Die Bedeutung der spinocerebellaren Systeme, Wiesbaden, 1907, Neurol. Centralbl., 1912, p. 270.

[4] Marburg: Arch. f. Physiol., suppl., 1904, p. 457.

[5] Bruce and Schäfer: Quart. Jour. Exper. Physiol., 1910, No. 3.

but, unlike Bing, could not demonstrate any sign of ataxia, though paralysis did occur. On the other hand, Lewandowsky,[6] Rothmann[7] and others rather positively state that section of Gowers' tract in dogs does produce a certain degree of analgesia.

In the following experiments our object was to completely destroy the anterolateral region, including Gowers' tract, and not to injure the area occupied by the dorsal cerebellar tract or any part of the gray matter. Dogs were chosen for purely extraneous reasons.

Under complete ether anesthesia an incision was made over two spinous processes and carried down directly to the bone; this incision was carried on as a subperiosteal resection of the processes and the laminæ of the vertebræ, until the cord was exposed for about 3 cm. The dura was slit longitudinally in the midline and retracted on either side; the cord was then rolled over as gently as possible till the line of insertion of the ligamentum denticulatum was identified, believing this to correspond quite accurately to the posterior limits of the anterolateral tract; a small knife was inserted immediately in front of that point and thrust obliquely forward, then cutting outward through the periphery of the cord. A similar incision was made at the corresponding point on the opposite side. There was little hemorrhage. The dura was closed with a fine silk suture and the wound repaired with catgut. Iodin and collodion were used for dressing. Wound healing was perfectly normal.

The dogs were carefully watched from day to day; generally the first observations recorded were made a day or two after operation, so that the effects of the operation itself might not be mistaken. Here we wish to point out one possible source of confusion. Any one at all familiar with animals, particularly the laboratory dog, should not lose sight of the fact that their general conduct and intelligence influences the manner in which they react to stimuli of any kind; even in health this may differ very greatly. Each dog has his own peculiarity, and has a distinct individuality not unlike man. Many are extremely dull and apathetic and others highstrung, intelligent and active, and accustomed to responding quickly to the various stimuli originating through the association of friends and master. No doubt it was a thorough appreciation of these facts which led Lewandowsky and Kallisher to adopt their methods of training. Our dogs were not trained; nevertheless a careful estimate of individual traits and temperament was constantly kept in mind. In considering disturbances of sensation these facts are of the utmost importance and have therefore been referred to in detail.

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[6] Lewandowsky: Handbuch der Neurologie, pp. 773 and 779.

[7] Rothmann: Berl. klin. Wchnschr., 1906, No. 2, p. 47; ibid., 1901; Arch. f. Physiol., 1902, p. 154; Suppl., 1902, p. 440; Ztschr. f. klin. Med., 1902, xliv, 183; Neurol. Centralbl., 1911, p. 1207.

After a period of time the dogs were killed and the brains and cords immediately removed and placed in Müller's fluid. The segment in which the incision had been made was mounted in celloidin and cut in serial sections in order to ascertain the exact limits of the area destroyed; sections were also made from each segment of the cord and parts of the medulla and pons, so that the course of degeneration could be followed.

In the following descriptions of cases the data have been given chronologically so as to show the course of the general symptomatology; then follows the microscopic study.

DOG 1.—Black and tan, short-haired male dog, weighing about 25 pounds. Operation Dec. 4, 1911. Cutting the anterolateral column of both sides in the seventh thoracic segment.

December 5: Incomplete weakness of both hind legs, more marked on right. Dog can stand but often falls behind. After falling it can rise on hind legs without evidence of weakness, but is awkward and can only stand a second or two before again falling. It frequently assumes curious positions as if kneeling, or may stand with right foot flexed so that dorsum of foot touches the floor; sometimes sits with weight on one leg which is crossed under rump. These positions do not seem to cause any discomfort, and appear to be from impairment of the sense of muscular position; it is very striking, and it even seems doubtful if there is any real motor weakness, the apparent weakness being due to uncertainty of ataxia. Sensation for pain tested with pin point and with hemostats shows definite impairment; dog reacts quickly in front legs and shoulders, and very slowly or not at all on hind legs. Extreme heat gives the same results.

December 8: Sensory tests gave same results. Ataxia seems definite and very striking, no motor palsy.

December 10: Sensory tests the same. Dog can stand on all four limbs without evidence of ataxia or of motor weakness. Ataxia very striking when in motion, trunk and rump involved, feet frequently crossed and seem to be misplaced so that dog often falls; curious positions when at rest as before.

very striking. Both sides alike, possibly right side more ataxic.

Jan. 4, 1912: Bilateral ataxia of hind legs and rump very distinct in walking and running. Dog falls very frequently with hind leg. Curious position of legs seen less often than before. Painful faradic current causes slight or doubtful evidence of discomfort in hind legs and rump, but distinctly less marked than on front legs where it is positively painful. When applied to soles of feet, hind legs are drawn away but not promptly, while on soles of front feet legs are drawn away quickly and with vigor. Application of hemostats caudal to site of operation is distinctly less painful than above operation-scar, though when attention is directed to the procedure, the dog seems to feel some discomfort very occasionally. For extreme heat the same holds true.

January 11: Ataxia is gradually recovering; no other change.

January 18: The same as on the 11th.

February 1: All sensory tests the same as before, but at times very confusing, depending very much on whether or not dog's attention is directed to what examiner is doing or to distant object. When attention is held by assistant then analgesia is very pronounced and unmistakable, but when attention is not diverted, sensation is affected, but not at all pronounced or even doubtful. Rapid examination might easily show that sensation was normal, but careful, frequent and prolonged examinations make it unquestionably correct that there is incomplete analgesia and thermo-anesthesia and pronounced bilateral ataxia of hind legs and no motor paralysis.

Dog 2.—Black and tan, long-haired male dog, weighing about 25 pounds. Operation Dec. 5, 1911: cutting the anterolateral column of both sides at seventh thoracic segment.

December 6: Dog cannot stand or walk on account of inability to use hind legs. Tendon-reflexes are equal and active. There is no spasticity nor do the hind legs feel exactly flaccid. Two sides are alike. Sphincters are not affected; no spontaneous pain. Application of hemostats over hind legs and also heated instrument seems to cause no discomfort; when it is applied to front legs dog reacts rather slowly so that conclusions are unreliable.

December 10: Ability to use hind legs voluntarily rapidly returning. Dog makes effort to stand and walk: can rise on hind legs but generally falls; muscular weakness very doubtful. Seems as if he had some loss of sense of muscular position; legs appear awkward. Pinching of skin over hind legs with hemostats does not seem to be appreciated; but when applied to front legs it is quickly recognized. Testing with heat perhaps the same but unreliable.

December 15: Loss of the sense of muscular position or ataxia very striking. No motor weakness. When standing at rest dog is inclined to lower head below line of shoulders, with hind legs somewhat further apart than normal and slightly rotated outward, vertebral column shows slight tendency to arching in lower thoracic and lumbar region; this varies from time to time; the tail is held in what may be a purposeful position, straight out from the body, varying in its angle as position of legs is changed, but generally curved, tip turned downward or laterally and more or less rigid at base. The general position is very like that assumed by a dog standing on a swing in motion, attempting to retain balance. In walking or running he repeatedly falls with hind legs, feet are often crossed but when moved are lifted clear of the floor, though not higher than normal. Rump sways a little. Dog often assumes unnatural positions, typical kneeling position, or stands on dorsum of foot, toes being in flexion, one or both hind legs may be crossed under rump when in sitting position. This does not seem to be in the least uncomfortable.

Pinching with hemostats and application of extreme heat over rump, hind legs and belly do not appear to be as distinctly or as quickly felt as when front legs are tested. At times there seems to be very decided analgesia but a few minutes later dog seems to feel quite well; although when compared to front legs there is a decided difference in reaction time. The tendon-reflexes are equal and active.

December 20: About the same as on the 15th.

December 29: Ataxia is somewhat less marked. Dog does not fall so often in walking and running. Sensation the same as before.

Jan. 4, 1912: Much the same as before. Application of painful faradic current gives exactly the same results as in first dog.

January 15: Sensory tests about the same. Analgesia seems definite at times while at another examination it seems very doubtful. Ataxia less marked; dog still falls when walking, but unnatural positions are assumed only very occasionally. In standing the position already described is still very striking.

January 24: Just the same.

February 6: Dog killed sixty-three days after the operation and the brain and cord removed for examination.

Conclusions: Ataxia very striking.

Impairment of sense of pain and for extreme heat and painful faradic current over lower half of body and lower extremities.

Microscopic examination showed that the anterior and lateral portions of the white columns of the cord had been destroyed on both sides. A line drawn through the posterior limits of this area would just bisect the central canal. Anteriorly it extended to a point which corresponded to the position of the most medially situated anterior root fibers. The ventral and dorsal cerebellar tracts were degenerated as far as the pons. In addition some of the degenerated fibers seen in the thoracic segments of the cord corresponded to the positions given by Edinger[8] for the spinotectal, spinothalamic, vestibulospinal and spinovestibular fibers. In segments below the area destroyed there were a few scattered degenerated fibers situated in the extreme lateral periphery. The pyramidal tract and von Monakow's bundles did not seem to be injured. The posterior columns were intact.

In both dogs motor paralysis was noted immediately following the operations, but it disappeared rapidly and was replaced by very marked ataxia. Why motor weakness should occur at all is difficult to explain, for we believe that Gowers' tract is sensory in function and the microscopic examination showed that the pyramidal and von Monakow's bundles were not injured. It may have been from temporarily disturbed vascular supply.

Rothmann has proved that when both the pyramidal tracts and von Monakow's bundles are destroyed, either in dogs or in monkeys, the resulting paralysis may not be permanent. He was forced therefore to conclude that volitional impulses might be conducted from the cortex by some other route. In this connection Schäfer's experiments are important. He cut Gowers' tract in monkeys and obtained motor weakness but without any evidence of ataxia or of cutaneous sensory disturbances. In order to explain the occurrence of paralysis he inclined to the opinion that voluntary impulses must be carried from the cortex down the cord by fibers which arise in Deiter's nucleus, and pass through the ventral and anterolateral columns near the periphery, and represent therefore a subsidiary motor pathway. It does not seem possible to make a complete section of the anterolateral tracts without destroying these long descending fibers; in our cases they must have been destroyed, yet motor paralysis was not permanent. Microscopic examinations of the segments below the level of section did reveal some evidence of scattered descending degeneration; the great majority of degenerations, however, occurred above the area destroyed and extended in the ascending direction.

Consequently Schäfer's[9] explanation does not seem to us entirely satisfactory. The experiments of Mott and

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[8] Edinger: Bau der nervosen Zentralorgane, Ed. 8, 1911.
[9] Schäfer: Jour. Physiol., 1899, xxiv, 23.

Sherrington,[10] on the other hand, are most suggestive. They divided the posterior roots of the nerves supplying the brachial plexus in a monkey and found that the arm was as much paralyzed for ordinary volitional movements as in other cases in which the motor nerves themselves were cut. This they attributed to the cutting off of sensory impulses which pass from the muscles to the cortex. Bastian, referring to these experiments, suggested that in consequence of dividing the posterior roots there must have been a loss of tone in the neuromuscular apparatus. This seems to us the most probable explanation, for we know that the fibers of Gowers' tract represent the secondary continuations of some of the posterior root fibers, and it is probable that the function of one is represented in part by the other. Furthermore, it is known that the fibers of Gowers' tract end chiefly in the cerebellum.

Inasmuch as our dogs did not show the ordinary signs of spastic paralysis and the apparent motor weakness gradually subsided, and ataxia became more and more apparent, and finally entirely replaced disturbance of voluntary motion, we believe that motor paralysis in the true sense never existed. But on account of a very profound derangement of the neuromuscular apparatus, together with impairment of cutaneous sensibility, a pseudoparalytic state or a true paralytic ataxia did occur, because sensory impressions which should pass through Gowers' tract had been cut off; and as the compensatory function of the remaining sensory tracts was being established the apparent paralysis gradually disappeared and ataxia which had previously been masked then became evident and remained permanent.

The character of the ataxia was similar to that described by Bing and Marburg and, according to them, should be expected, as the spinocerebellar tracts are essentially the afferent connections between the cerebellum and spinal cord. It seems to us that the rubrospinal and vestibulospinal fibers may, as Bing has lately indicated, repressed efferent connections between the cerebellum and cord and their destruction might be partially responsible for the extreme incoordination; however, proof is lacking.

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[10] Mott and Sherrington: Quoted in Schäfer's article.

In regard to the occurrence of cutaneous sensory disturbances there is considerable difference of opinion. Schäfer, Bruce, Bing and others were unable to demonstrate definite cutaneous disturbances. On the other hand, Rothmann, Schuster,[11] Lewandowsky and others all agree that Gowers' tract in the dog does convey painful sensations, though the posterior columns are also concerned. Why such contradictory views should be expressed we have no explanation to offer unless it is that the character of the dogs has not always been considered.

In concluding we feel certain that our dogs did have a definite, though incomplete, loss of cutaneous sensation for pain and for extreme heat, and very pronounced ataxia of the hind legs and rump. The fibers whose function it is to conduct painful sensations probably are connected with the thalamus and are situated close to the tractus spinocerebellaris ventralis, whose function is chiefly concerned in the regulation of purposive movements.

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TUBERCULOUS MENINGITIS

A PATHOLOGIC REPORT OF NINE CASES *

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I recently had the opportunity of examining histologically nine cases of meningitis, seven (Cases 1, 2, 3, 4, 5, 6 and 7) of which had been diagnosed clinically and pathologically as tuberculous meningitis. Case 8 had been diagnosed as one of tuberculous meningitis from which the patient recovered only to succumb a month later to an attack of pneumonia.

The findings are interesting as showing the histologic features in a recovered case of meningitis.

Case 9 was diagnosed clinically and pathologically as pneumococcus meningitis and is of interest in comparison with the findings in the tuberculous cases.

As far as possible the material was taken from those regions of the cortex where the tubercles were not found. While all the sections were stained for the tubercle bacillus, none was discovered in any case after careful search. It should be mentioned that some of the specimens were hardened in Kaiserling, which may have affected its staining properties for the tubercle bacillus. The results were interesting and instructive and, though they do not permit the drawing of any positive conclusions, they suggested some ideas which are worthy of mention.

It seemed possible that the lesion which was fairly uniform in its histologic character was caused not alone by the tubercle bacillus, for reasons which will be developed later on in this paper. It further seemed possible that the meningitis was due to a mixed infec-

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tion. If this could be proved it would seem but a step further to obtain a vaccine which might operate against such a process. If this could be successfully combated, there then would remain the tuberculous process alone, which in itself may be looked on as less fatal than the mixed infection, and within the possibility of cure.

Experiments are now on foot, in conjunction with Dr. Herbert Fox, with this end in view and a report will be made later, embodying the results of these experimentations.

I am indebted to Dr. J. Crozer Griffith, Dr. J. C. Gittings and Dr. Alfred Hand, for the privilege of reporting some of these cases, and Dr. C. Y. White for the pathologic material from these cases from the Children's Hospital.

Case 1.—*Patient.*—A. P., aged 12, was admitted to the Children's Hospital Sept. 14, 1905. The family and personal histories were negative. The symptoms dated from the day before admission and consisted of fever, delirium, persistent vomiting, headache, drowsiness and slight stiffness of the neck. On admission the abdomen was distended and there was obstinate constipation. Examination of the lungs was negative and also of the heart except that it was rapid. The pupils were dilated and equal. Kernig's sign was present. The head was retracted and *tache cérébrale* was demonstrated. Numerous dark-brown spots scattered over the body were seen. Later the legs became rigid, the face was drawn to the right, and deafness ensued. Examination of the eyes, by Dr. William Campbell Posey, was negative, except that the pupils were dilated.

An irregular temperature marked the course of the disease ranging from subnormal to 104 3/5. The patient was most of the time in a semistuporous state, and died November 26.

Autopsy.—The dura was opaque and thickened. The pia-arachnoid was also opaque and thickened, and areas of exudate were observed on the base, the convexity and along the sylvian fissure. A few miliary tubercles were observed. No tuberculous process was seen elsewhere.

Microscopic Study.—The pia was the seat of a round cell infiltration, the cells being mainly of connective-tissue type, though there were some mononuclear round cells. Connective-tissue formation enclosing blood-pigment and numerous blood-cells was also observed. The connective-tissue cells showed in places vacuolization and some contained blood-pigment. The intima of the arteries and veins was thickened and showed proliferation of the connective-tissue cells. The pia of the medulla was the seat of a marked round cell infiltration, the cells consisting of small mononuclear cells and large polynuclear cells. In the cortex there was a marked degree of perivascular dilatation but the cortical cells stained well.

This case was of the mixed type, the proliferative type predominating in the pia of the cerebral cortex, and the exudative type in the pia of the medulla. This case was clinically one of tuberculous meningitis and while no tubercle bacillus was found in this or, indeed, in any of the cases of this series, microscopically it coincided with the usual histologic characteristics of this disease.

Case 2.—*Patient.*—M. H., aged 13 months, was admitted to the Children's Hospital March 16, 1906. Histories, family and personal, were negative. The patient died March 25, seven weeks after the onset of the disease, though the nervous symptoms preceded death but three weeks. The first symptom consisted of diarrhea with green stools. A month later the child was seized with convulsions affecting the left side of the face, arm and leg. The eyes were turned toward the right and later the right side of the face, the right hand and foot were convulsed. The movements were more or less continuous and there was some general rigidity which later became very marked, the legs and arms being held in extension. Kernig's sign was not clearly demonstrated. The knee-jerks were not increased. Lumbar puncture was done without effect on the symptoms. The heart was rapid and there was some dyspnea. There were no spots on the body and examination in other respects was negative. The temperature ranged from 99.2 to 104.4.

Autopsy.—There was found general tuberculosis involving the lungs, epicardium, peritoneum, kidneys, stomach, intestines and lymph-glands.

Microscopic Examination.—The infiltration of the pia consisted mainly of cells of the connective-tissue type. Many plasma cells, however, were present. Numerous red blood-cells were found in the thickened pia. The veins were slightly thickened, the intima showing a marked proliferation of the connective-tissue cells. There was a slight perivascular distention and slight perivascular infiltration of the cortex.

In this case in which the nervous symptoms manifested themselves for three weeks only before death, there is evidence clinically of an acute process. The type of cellular infiltration is strikingly proliferative. The presence of blood in the process is worthy of mention as being present in both the exudative and proliferative forms of tuberculous meningitis.

Case 3.—*Patient.*—R. S., aged 6½ years, was admitted Nov. 14, 1906, to the Children's Hospital and died November 28. The family and personal histories were negative. Duration of the disease was twenty-two days and began with loss of appetite, headache. slight enlargement of the tonsils and fever. On admission the examination of the lungs and heart, abdomen and spleen was negative. There was marked *tache cérébrale.* The patient was irritable and lay curled up on one side. There developed then rigidity of the neck, dysphagia and general convulsions. Kernig's sign was present on the sixth day. The patellar reflexes were irregular, being sluggish on the right. Paralysis of the left external rectus was present associated with nystagmus. Babinski phenomenon was present and finally Cheyne-Stokes respiration.

Autopsy.—A diagnosis of tuberculous meningitis was made, though no note of any tubercles was made at the time of the necropsy.

Microscopic Examination.—The pia showed the presence of a dense infiltration which contained many mononuclear and polynuclear leukocytes. In other places the cellular infiltration consisted largely of endothelial cells which was very marked around the blood-vessels. Red blood-cells were numerous and a number of plasma cells were observed. Where the tissue was loose the cellular infiltration was composed largely of lymphocytes and leukocytes, but where the tissue was denser the connective-tissue cells predominated. The blood-vessels showed some thickening of the walls, and in places accumulation of round cells beneath the intima was pronounced.

In the cortex there was perivascular distention and slight round cell infiltration around the blood-vessels. Marked encephalitis was observed, the leukocytes predominating in the exudate.

This case illustrates the mixed form, showing the presence of both the exudative and proliferative type of tuberculous meningitis.

Case 4.—*Patient.*—M. E., aged 19 months, was admitted to the Children's Hospital April 24, 1906, and died two days after admission. Her mother had died of tuberculosis four months previously, but otherwise the family history was negative. The patient had suffered from a cough and general irritability for four months previously.

Two days before admission she became first dull, then unconscious. She was able to move the left side of the face only and the eyes twitched at times. There was general rigidity, more on the right side. The left eyeball was drawn inward and there was also palsy of the right external rectus. Kernig's sign was not definite.

In the lungs coarse moist and fine dry râles were audible. The spleen was palpable and the liver enlarged. Examination of the heart was negative. The temperature ranged from 98 to 102.8.

Autopsy.—A diagnosis of general tuberculosis was made, involving the lungs, liver, spleen, kidneys, intestines and meninges. The exudate over the meninges occupied the posterior and superior surface of the cortex posterior to the middle line. The base of the brain was also the seat of infiltration.

Microscopic Examination.—In the pia the infiltration was made up of leukocytes and lymphocytes mainly. In places there was present in the pia large quantities of red blood-cells. The process was mainly of an exudative type. The cortex was invaded in places, the cells in the subcortical layers being of the mononuclear type and containing darkly-staining nuclei, but polynuclear cells and some plasma cells were also observed. Perivascular distention of the cortex and round cell infiltration around the vessels were present. Obliteration of the small vessels with some slight proliferation of the intima in others was noted.

This is an example of the purely exudate type of meningitis. The case was a rapid one, the evidence of meningeal implication lasting but four days.

Case 5.—*Patient.*—H. P., aged 2 years, was admitted to the Children's Hospital March 25, 1906, and died March 31. The family and previous histories were negative except that one sister died in spasms. There was no history of tuberculosis. The child had been sick for two weeks before admission, making the duration of the disease eighty-one days. The disease was ushered in with vomiting, constipation, slight cough and somnolence.

On admission the child was unconscious, the eyes were open, the pupils large, reacting sluggishly, strabismus was present and there was rigidity of the neck and extremities. The knee-jerks could not be developed, though ankle-clonus and the Babinski phenomenon were present. Kernig's sign was positive. The temperature ranged from 98 to 100 until just before death when it rose to 105.2.

Autopsy.—A diagnosis of tuberculous meningitis was made and miliary tuberculosis of the lungs, liver, kidneys, spleen, intestines and bronchial glands was present. There was an exudate over the motor region on the right side and also at the base.

Microscopic Study.—The pia was thickened, this being due to fibrous tissue containing many connective-tissue cells and a few round cells. Some red blood-cells were found in the thickened pia. The veins were surrounded by many connective-tissue nuclei, but the blood-vessels themselves were not implicated.

The cortex was invaded by the process. There was some perivascular dilatation and the blood-vessels were distended and showed slight proliferation of the intima.

The proliferation type of tuberculous meningitis predominated in this case. The process was not extensive or very intense, and was evidently, as the clinical story would indicate, a chronic process.

Case 6.—*Patient.*—B. P., aged 3½ years, was admitted to the Howard Hospital Jan. 12, 1908, and died January 13. The family and previous histories were not obtainable. Patient on admission showed paralysis of the right side of the face, right arm and leg. The right arm was flexed and was distinctly spastic. The left arm and leg was kept in constant motion. Movements of the head were painful and disclosed stiffness of the neck muscles. The right biceps-jerk was increased but the left could not be elicited. Both knee-jerks were increased and equally so. Babinski phenomenon was present on both sides but ankle-clonus was absent. The pupils reacted normally. Temperature ranged from 102 to 105.

Autopsy.—This revealed the presence of tuberculous meningitis and hydrocephalus. Miliary tuberculosis of the kidney and peritoneum was observed.

Microscopic Study.—The pia from the paracentral region was the seat of a dense process which implicated both the pia and the superficial cortical layers. Connective-tissue cells predominated, though there were some trifoil cells but these were mainly perivascular and were present where the infiltrative lesion was less dense. The connective-tissue cells were found in various states of degeneration many of them showing vacuolization. In the superficial layers of the diseased pia the loose connective tissue was infiltrated with mononuclear and polynuclear cells, and a few plasma cells, and was also the seat of extensive extravasation of blood.

In the frontal region the same condition obtained. In this region the blood-vessels were diseased, the outer walls being the seat of cellular accumulations. The cortex had been invaded slightly in places, the tissue being loose and the seat of some space formation. Just below the pia there was present cellular infiltration, some cells being mononuclear round cells, some being pale and granular without a demonstrable nucleus, some elongated cells, some cells with two nuclei, some trifoil cells and some large cells with many nuclear fragments tending to undergo vacuolization. Phagocytosis was also observed in the subcortical layers.

The cortex of the cerebellum was the seat of a marked encephalitis. The pia of the cord was the seat of a mononuclear round cell infiltration of moderate intensity and the posterior roots were infiltrated with cells of similar nature. The anterior roots were similarly affected, but to a less degree.

This case belonged to the proliferative type of tuberculous meningitis, but the characteristics of the exudate type of the disease was also present to a marked extent. and it therefore more properly comes under the head of the mixed type.

Case 7.—*Patient.*—V. S., aged 2 years, was admitted to the Howard Hospital Nov. 4, 1907, and died November 8. The family and previous histories were unobtainable. The child was admitted with tumor of the abdomen which proved to be a distended bladder.

Examination.—The patient was semicomatose on admission. The pupils did not react to light and the eyeballs were moved from side to side constantly there being a tendency to left conjugate deviation, with transient weakness of the left internal rectus. The scalp and the nape of the neck were tender to pressure and the head was retracted. There was general rigidity of the limbs and from time to time a clonic spasm of both arms. *Tache cérébrale* was present. The knee-jerks could not be elicited, probably due to the rigidity, and the Babinski phenomenon was observed on both sides. The examination of the eye-grounds, by Dr. William Campbell Posey, was negative.

Autopsy—General tuberculosis was found of the lungs, liver, mesenteric glands and also the meninges, though microscopically no tubercles could be observed in the process in the meninges.

Microscopic Study.—The pia was much thickened and contained leukocytes and connective-tissue cells. The cells were mainly of the connective-tissue type, though many plasma cells were present and some red blood-corpuscles. The blood-vessels showed marked cellular proliferation of the walls. Around the blood-vessels there was marked cellular accumulation of connective-tissue cells. The cortex was the seat of encephalitis in places and there was perivascular distention, the cortex being in places rarified.

This case belongs to the proliferative type of tuberculous meningitis.

Case 8.—*Patient.*—J. R., aged 6, was admitted to the Children's Hospital Jan. 27, 1906, and died January 30. The family and previous histories were entirely negative. She had previously been treated at the Children's Hospital for an attack of meningitis, which lasted from Dec. 1, 1905, to December 28, when she was discharged as cured, save for paralysis of the external rectus muscle of the left eye.

At that time she had suffered with rigidity of the cervical muscles and of the legs which were held in extension. Kernig's sign was present and there was paralysis of the external rectus of the left eye, which remained paralysed. The knee-jerks were absent and there was no ankle-clonus or Babinski phenomenon present. The pulse was irregular and intermittent. The temperature was not characteristic and ranged from 97 to 104. Examination of the fluid obtained by lumbar puncture showed the presence of intercellular diplococcus and an excess of polymorphonuclear cells. A month after her discharge she returned with symptoms of lobar pneumonia from which she died in a few days.

Autopsy.—The pathologic diagnosis was double lobar pneumonia and acute fibrinous pleurisy. The lung showed some cavity formation and pulmonary tuberculosis was probably present, including some miliary tubercles. Arachnoid was congested except over the frontal lobes.

Microscopic Examination.—The infiltration in the pia was not intensely cellular. There were some leukocytes in small quantities, numerous round cells, numerous connective-tissue cells and some large round mononuclear cells. Many red blood-cells were seen. Some of the connective-tissue cells showed advanced vacuolization and some contained blood-pigment. Other cells stained deeply and contained dark round nuclei. The vessels of the cortex were filled with red blood-cells, the walls were thickened, showing hyaline degeneration and some of the vessels were obliterated. There was perivascular round-cell infiltration of the cortex which was rarified. Fresh hemorrhages were found in the subcortical layers which was also the seat of a slight cellular infiltration.

In this case a diagnosis of tuberculous meningitis had been made, from which the patient made a recovery, having been discharged as cured. It may have been, and probably was, a diplococcus meningitis, though the presence of cavity formation in the lungs leaves some doubt as to whether or not it was not actually a case of tuberculous meningitis after all. This case is interesting as illustrating the alleged cures of tuberculous meningitis, about the exact nature of which, however, it seems to me there is some doubt.

The microscopic findings were similar to those found in tuberculous meningitis of a proliferative type but less marked. Added to this there was evidence of an old process in the fibrous nature of the process and in the presence of degenerated cells.

The cortical lesion was of two kinds, an old process, represented by the rarification of the cortex and degenerated blood-vessels, and a recent process characterized by fresh hemorrhages and some round cell infiltration, perivascular in type.

The older process, I take it, was the remains of the diseased condition from which the child recovered, apparently, a month prior to the onset of the fatal pneumonia. The more recent process could be explained by a meningeal extension of the pneumococcus invasion.

Case 9.—*Patient.*—J. S., aged 1 year, admitted to the Children's Hospital September 26, 1907, died October 21. The family and previous histories were entirely negative. The child was breast-fed from birth. The duration of illness was thirty days and began with high fever and vomiting. Shortly after the onset the patient was comatose, the neck became stiff, the pupils were dilated, reacting sluggishly to light, and the reflexes became increased. Except for two vesicles which were found on the cheeks, no eruption was observed.

Later there developed internal strabismus, tremor of the head and body, spasm of the hamstring muscles and Cheyne-Stokes breathing.

On several occasions lumbar puncture was made; the fluid contained, besides pneumococci, many polynuclear cells, leukocytes and red blood-cells. The temperature ranged from 98.2 to 105, rising finally to 107.2 before death.

Autopsy.—Cerebrospinal meningitis was diagnosed. None of the other organs showed any tuberculous involvement. There was marked distention of the fourth ventricle.

Microscopic Examination.—The pia was fibrous and sparsely dotted with cells, mononuclear in type. The nuclei stained poorly in the outer layers of the thickened pia and in some the protoplasm was granular while in others there were two nuclei present. In the inner layers there were many deeply staining, small and irregular nuclei. A few plasma cells were observed. Around the blood-vessels the cellular accumulation was marked, composed mainly of the connective-tissue type of cell. There was also a slight proliferation of the intima of the small veins. In the cortex capillary hemorrhages were observed and there was some perivascular distention. In some of the cells four nuclei were observed, suggesting karyokinesis.

This case was diagnosed as pneumococcus meningitis, the pneumococcus having been found in the cerebrospinal fluid. It is of the mixed type showing proliferative and exudative characteristics, and it is to be noted that microscopically it differed in no way from the usual picture of tuberculous meningitis.

SUMMARY

Of these cases only one (Case 4) properly belongs to the purely exudative type of tuberculous meningitis. Three (Nos. 2, 5 and 7) are proliferative cases and three (Nos. 1, 3 and 6) are of the mixed type. Blood or blood pigment was found in eight (Nos. 1, 2, 3, 4, 5, 6, 7 and 8) of the cases. Plasma cells were found in six of the cases(Nos. 2, 3, 4, 6, 7 and 9).

Blood-vessel changes to a more or less degree were present in all the cases. Cortical changes were present in all the cases, in three of which marked evidence of encephalitis was observed. The duration of the disease seems to have little if any bearing on the character of the exudate. The duration of the disease in the exudative type was four days; in the proliferative type three weeks, twenty-seven days, eighty-one days and one of short duration; in the mixed type seventy-four days; thirty days, twenty-two days and one of short duration.

In this study an effort was made to investigate the histologic characteristics of the process at those places removed from the tubercles themselves. The lesion in all cases was more or less similar, except in the one case showing a purely exudative type and this was, beyond doubt, one of typical tuberculous meningitis.

Cases 8 and 9 are doubtful cases of tuberculous meningitis but the histologic findings are identical to those found in the other cases.

This brings up the question as to the nature of so-called tuberculous meningitis. I believe that it is possible, if not probable, that the meningeal exudate in these cases is due to a mixed infection and not alone, or at all, to the toxins of the tubercle bacillus.

The rapidity of the course of the disease in these cases, which from its inception till the death of the patient is often but a very few days, is unlike the slow chronic process characteristic of tuberculosis in other organs. Moreover, in cases of tuberculosis of the meninges of patients who succumb to this disease in other organs, the histologic picture is not the usual one found in the tuberculous meningitis of children.

The process in tuberculous meningitis, I believe, does not necessarily originate solely from the tubercle bacillus or its toxins. The absence of the bacillus of Koch, which is not unusual, speaks against it. Moreover, the histologic picture of the lesion removed from the tubercle is of a round cell infiltration which, I believe, cannot be said to be pathognomonic of a tuberculous process.

Non-specific or simple inflammatory reactions in tuberculous meningitis have been already described by Villaret and Tixier,[1] Peron,[2] Hayen,[3] Chantemesse[4] and others.

Diffuse leukocytic infiltration without tuberculous granulations and without definite tubercles and giant cells has also been observed by Siredey and Tinel.[5]

That it may be a mixed infection is well illustrated in the observations of Perrin,[6] who found the diplococci

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1. Villaret and Tixier: Compt. rend. Soc. de biol., 1905, p. 660.
2. Peron: Arch. gén. de méd., 1898, p. 413.
3. Hayen: In Josué and Salomon (Note 16).
4. Chantemesse: In Josué and Salomon (Note 16).
5. Siredey and Tinel: Lancet, London, April, 1907, p. 1033.
6. Perrin: Rev. méd. de l'est., 1902, No. 34, p. 677.

in the cerebrospinal fluid; of Kneass, Hendrickson and Sailer,[7] who isolated the *Micrococcus tetragenus* in the spinal fluid, as well as the tubercle bacilli in the smears from the tubercles in the meninges; of Griffon and Abrami,[8] in whose case the cerebrospinal fluid was invaded by the *B. coli;* of Marotte,[9] who described a case of septicemia of tetragenous origin coincident with tuberculous meningitis; and of Poisseau and Tixier,[10] in whose case were found diplococci not taking the Gram stain.

In two cases in my series, moreover, the diplococcus was found in the cerebrospinal fluid. In this connection, however, should be mentioned the contention of Sicard,[11] that the exudate in the pia is not the result of polymicrobic infection and that the bacillus of Koch and its toxins are capable in themselves of creating these granulations.

The experimental evidence is somewhat conflicting. Hektoen[12] failed to produce typical meningitis by injecting tubercles into the carotids of rabbits, though miliary tubercles were observed in the meninges.

On the other hand, Sicard produced experimentally a diffuse process consisting of a leukocytic infiltration and other lesions similar to the diffuse meningeal changes found in man. There is no evidence in these experiments to exclude, however, the existence of a mixed infection.

At the same time the experiments of Martin and Vandremer,[13] and Peron,[14] show, according to these observers, that the toxins of tuberculosis play an important rôle in the development of tuberculous meningitis.

CELLULAR CHANGES

The cellular changes have been studied by Diamond,[15] Josué and Salomon,[16] Poisseau and Tixier,[10] Higgs[17] and Siredey and Tinel.[5]

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[7] Kneass, Hendrickson and Sailer: Jour. Nerv. and Ment. Dis., 1903, p. 431.

[8] Griffon and Abrami: Bull. et mém. Soc. méd. d. hôp. de Paris, 1906, xxiii, 613.

[9] Marotte: Bull. méd., 1909, p. 259.

[10] Poisseau and Tixier: Gaz. d. hôp., 1909, p. 979.

[11] Sicard: Presse méd., 1900, p. 67.

[12] Hektoen: Tr. Chicago Path. Soc., 1894-5, i, 69.

[13] Martin and Vandremer: Compt. rend. Soc. de biol., 1898, p. 273.

[14] Peron: Arch. gén. de méd., 1898, ii, 412.

[15] Diamond: Am. Jour. Med. Sc., New Series, 1903, cxxvi, 147.

[16] Josué and Salomon: Bull. et mém. Soc. méd. d. hôp de Paris, 1903, xx, 1133.

[17] Higgs: Brit. Med. Jour., 1909, p. 1170.

Diamond[15] found that the plasma cells, lymphoid cells, and phagocytic cells formed the principal cell-content of the infiltration of vascular leptomeningitis. He claims to have been the first to have described the presence of the plasma cell in acute tuberculous inflammation. The phagocytes were looked on as transmitting the tubercles from one area to another.

Lymphocytes and polynuclear cells predominated in the cases of tuberculous meningitis in adults studied by Josué and Salomon.[16]

In the case cited by Poisseau and Tixier[10] the cellular infiltration was composed of mononuclear lymphocytes and endothelial cells without giant cells and finally, in the case of Siredey and Tinel,[5] the examination showed a diffuse leukocytic infiltration without tuberculous granulations or giant cells.

The exudate in Higgs' case consisted of fibrin and round cells, mainly large and small lymphocytes, but some polymorphonuclear leukocytes.

The cellular changes of the exudate of tuberculous meningitis are not absolutely characteristic, somewhat similar changes having been described by Spielmeyer,[18] as existing in trypanosomiasis and paresis (Gehry[19]), though Gehry concludes that the miliary foci in the pia give the process a typical appearance. While this is true, I think it still may be contended that the cellular makeup of the process remote from the tubercle is not typical of a tuberculous process.

VASCULAR CHANGES

The blood-vessels exhibited, in my cases, more or less change in seven of the nine cases studied. These changes consisted of thickening of the walls of the arterioles and some veins, which in one case went on to obliteration of the lumen of the small vessels. Perivascular round-cell infiltration was observed quite commonly. In some instances the coats of the veins were thickened and in others there was cellular proliferation of the intima. In one case an accumulation of round cells beneath the intima was observed; in another case cellular proliferation of the outer coat was demonstrated; and finally, in still another case the walls of the capillaries, in which the outer coat showed cellular proliferation, had undergone hyaline degeneration.

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[18] Spielmeyer: Die Trypanosomenkrankheiten, 1908.
[19] Gehry: Arch. f. Psychiat., 1909, p. 59.

The vascular changes described by Hektoen[12] which were present in all of his nine cases, consisting of a primary endarteritis and a phlebitis causing thrombosis and obliteration, were not uniformily present in my cases.

Hektoen believes that this primary endarteritis is due to the implantation of the tubercle bacilli on the intima. He describes, (1) a diffuse form in which proliferation of the epithelioid cells occurs between the elastic lamina and the endothelium; (2) isolated miliary tubercle on the intima either singly or in conjunction with diffuse endarteritis, and (3) diffuse changes which proceed from periarterial foci.

He quotes the works of Huguenin, Huttenbrener, Cornil and Hirschberg, who observed tuberculous endarteritis which was looked on as primary and due to the implantation of the tubercle on the intima.

Rindfleisch, Ziegler, Birch-Hirschfeld, Lancereau, Baumgarten, and Guernieri and others believed that the bacilli were localized in the adventitia, and Guernieri and Baumgarten described an endarteritis which they regarded as secondary (Hektoen[12]).

Blood-vessel changes have been described by Raymond,[20] who found in the majority of the vessels of the pia an infiltration of the sheaths. Vascular changes have also been described by Diamond[15] and Lartat-Jacob and Sabareanu.[21] In Dreher's case[22] proliferation of the intima went on to obliteration of the lumen, and there was also perivascular infiltration.

Ettinger,[23] on the contrary, said that the absence of obliterating arteritis was striking and that the walls, and especially the external and middle coat, are a little thickened but uniformly so, and that the infiltration of the round cells is regular.

ENCEPHALITIS

A slight diffuse encephalitis is always present in these cases, according to Oppenheim.[24]

Distention of the perivascular spaces was noted in seven of the cases under discussion. In three cases there was marked round cell infiltration of the superficial layer of the cortex and in another capillary hemorrhage was found. The cortex was rarified in two cases and the cerebellum was the seat of a marked encephalitis in one case.

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[20] Raymond: Rev. de Med., 1886, p. 230.

[21] Lartat-Jacob and Sabareanu: Bull. et mém. Soc. anat. de Paris, 1904, p. 161.

[22] Dreher: Deutsch. Ztschr. f. Nervenh., 1899, p. 58.

[23] Ettinger: Compt. rend. Soc. de biol., 1896, p. 23.

[24] Oppenheim: Lehrbuch der Nervenheilkunde, 1905, p. 776.

In this connection the work of Lhermitte[25] is interesting. He described tuberculous meningo-encephalitis and believed it impossible to decide, in a great many cases based on the histologic findings, whether the condition was one of tuberculous encephalitis or a simple (banale) inflammatory reaction. He found in these cases that the plasma cell was present in the cortex.

PLASMA CELL

Studies of the plasma cell in tuberculous meningitis have not been made to any extent until within the last ten years. Wolf believed that they were regularly present in tuberculous meningitis and believed, furthermore, that degenerated plasma cells were found in all cases, though in varying amount, and raised the question whether these did not have some connection with regressive metamorphosis. Gehry[19] believed that the plasma cell played an important rôle in the regressive changes in the nerve-elements.

The rôle of the plasma cell in vascular tuberculous meningitis was described at length by Diamond,[15] who found them present in chronic and acute cases of tuberculous meningitis.

They were not found in all of the cases reported in this paper. In six cases, however, they were present to a greater or less extent.

As to the origin or significance of the plasma cell, this paper does not strictly pertain. Suffice to say that much has been written on the subject and whether the plasma cell originates from the lymphocyte, as Krompecher,[27] Marschalko,[28] Justi,[29] Joannovics,[30] Porcile,[31] Councilman,[32] Herbert,[33] Jadassohn[34] and Schottlander[35] claim, or from the endothelial cell, as is believed by Unna,[36] Ehrlich[37] and others, is still a matter of dispute.

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[25] Lhermitte: Rev. Neurol., 1910, p. 49.

[27] Krompecher: Beitr. z. path. Anat. u. z. allg. Path. (Zeigler's), 1898, xxiv, 164.

[28] Marschalko: Centralbl. f. allg. Path., 1899, p. 851.

[29] Justi: Virchow's Arch. f. path. Anat., 1897, cl, 197.

[30] Joannovics: Ztschr. f. Heilk., 1899, p. 159.

[31] Porcile: Beitr. z. path. Anat. u. z. allg. Path. (Ziegler's), 1904, p. 276.

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[32] Councilman: Jour. Exper. Med., 1898, p. 393.
[33] Herbert: Jour. Path. and Bacteriol., 1901, vii.
[34] Jadassohn: In Krompecher (Note 27).
[35] Schottlander: Ueber Eierstocks Tubercul., 1897, p. 81.
[36] Unna: In Krompecher (Note 27).
[37] Ehrlich: Virchow's Arch. f. path. Anat., 1904, clxxv, 198.

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SYPHILIS A POSSIBLE CAUSE OF SYSTEMIC DEGENERATION OF THE MOTOR TRACT.[1]

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Many years passed before syphilis was generally recognized as the chief cause, and possibly the only cause, of tabes dorsalis, and it is largely owing to Erb that this recognition has been obtained. There is no objection on a priori grounds in accepting syphilis as a possible cause of the various diseases of the motor system, as primary lateral sclerosis, amyotrophic lateral sclerosis, and progressive spinal muscular atrophy. That in parasyphilis we may have a degeneration of the afferent fibers of the cord, viz., of the posterior roots and of their continuation within the cord, is a fact accepted by most neurologists. Can we have a similar degeneration of the central motor tracts and of the cells in the anterior horns of the spinal cord, or a combination of this type with degeneration of the posterior root fibers, as another form of parasyphilis? This idea is comparatively new, and yet there is no reason why this condition should not occur. If we accept this possibility then the diseases under consideration can not be regarded as always abiotrophic, even though they may be in certain instances.

Syphilis we accept as a cause of tabes, and in many cases we find no other signs of syphilis in the spinal cord than the degeneration recognized as tabetic in type, *i. e.,* the syphilitic poison has affected only nerve fibers. In many cases, however, a dense or moderate round cell infiltration of the pia occurs, and the degeneration of the posterior columns is systemic in character, and differs in no way from that of tabes, yet we regard these cases as examples of the more acute manifestations of syphilis, and attribute the degeneration of the posterior columns and the round cell infiltration to syphilis. It is very questionable whether in many of these cases we can make the systemic degeneration secondary to meningitis. The common lesions of syphilis of the spinal cord are lymphocytic infiltration and thickening of the walls of the vessels, and yet the latter is not always present. The round cell infiltration in many instances probably causes no lesions, but is an index of the syphilitic poison.

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[1] Read at the Thirty-eighth Annual Meeting of the American Neurological Association, May 30, 31, and June 1, 1912.

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I have repeatedly found a degeneration of the pyramidal tracts of the spinal cord, apparently primary, with marked lymphocytic infiltration of the pia. It seems improbable that the lymphocytic infiltration causes this degeneration, but it is likely that both have a common cause, the syphilitic poison. If motor fibers are susceptible to the syphilitic poison, it is natural to infer that motor cells likewise are susceptible, and it is not unreasonable to suppose that cases may exist in which the syphilitic poison exerts its influence chiefly on the motor system and produces little lymphocytic infiltration. What evidence can we find in literature as to muscular atrophy resulting from syphilis?

Muscular atrophy with tabes has been observed repeatedly since Charcot and Pierret reported a case of this kind in 1871. A recent review of the literature has been given by C. M. H. Howell in a paper by him and H. H. Tooth, in which the authors report a case of tabes with muscular atrophy, but not of the Aran-Duchenne type. Howell says he has examined in detail six cases of progressive muscular atrophy, and in none of these was there any distinct evidence of syphilis either in the meninges or in the blood vessels. It is clear, he says, that direct evidence as to any causal connection between syphilis and primary cell degeneration is very difficult to establish. If syphilis be the cause of cell degeneration in such cases as these, and he does not deny the possibility of this, it seems rather strange to him that this type of case is not more commonly met with. Syphilitic disease of the central nervous system is common enough, yet these cases of muscular atrophy with syphilitic origin in his opinion are decidedly uncommon. In my experience the frequency of these cases is much greater apparently than in his, and without any special search I report several cases that have recently come under my observation.

Also from a histological viewpoint the cellular changes, Howell says, are not those usually associated with the action of any toxin. I am inclined to think that the chronic cellular change from a toxin may differ from the acute effects. The changes in the nerve cells of the spinal cord observed by Tooth and Howell[2] were diminution in the number of the cells in the ventral horn cells and lateral horn cells and Clarke's column of cells, and degeneration of the surviving cells. The latter were shrunken, distorted and often elongated, some were more globular than are normal cells, the Nissl granules had lost their definition and stained deeply, and the nucleus in some cells was displaced and stained more deeply than usual. Some cells presented a different picture; they showed chromatolysis and vacuolation. I should hesitate to say that alterations such as these could not be the result of a chronic disease like syphilis.

Marie and Leri[3] have considered syphilis in an etiological relation to progressive spinal muscular atrophy. Until the present time it has played a modest rôle in this respect. Aran, MacDowald, Thouvenet, Charcot have found syphilis in the history of amyotrophic patients; Hammond, Niepce, Fournier, Misserbi have observed the relation of cause and effect between the two disorders; Raymond described diffuse vascular meningo-myelitis in a syphilitic in whom the symptoms differed from those of Aran-Duchenne atrophy only in a few respects, viz., pain and paresthesia preceding the atrophy; Leri has found syphilis frequently in the etiology of spinal amyotrophy. Amyotrophy appears especially in adult age, and the male sex, and frequently Leri has found a period of 7 to 15 years between the syphilitic infection and the beginning of the atrophy.

In Dana's[4] 72 cases of progressive muscular atrophy 19 gave a history of syphilis, which would be about 25 per cent. In none of his three cases of amyotrophic lateral sclerosis was there a history of syphilis. In 33 cases, in which the symptoms began in the hand and arm, and were of the ordinary Aran-Duchenne type, there were 9 cases. Among the 14 cases associated with spastic symptoms there were 3 cases of syphilis, and in the bulbar or bulbo-cervical type, of which there were 11, there were 3 cases. In all types, therefore, whether beginning in the arms, legs, or muscles innervated from the medulla oblongata, the percentage of syphilis seems to be about the same.

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[2] Tooth and Howell, Proceedings of the Royal Society of Medicine, Neurological Section, Feb., 1912.

[3] Marie and Leri, Traité de Médecine, Bouchard and Brissaud, second edition, Vol. IX, p. 637.

[4] Dana, JOURNAL OF NERVOUS AND MENTAL DISEASE, 1906, p. 92.

Objections have been made by Tooth and Howell that such arguments as are here offered are of the post hoc order and prove nothing. They are, however, suggestive.

Without attempting to collect all the reports of syphilitic muscular atrophy I refer briefly to a few. Lannois[5] reported a clinical case of muscular atrophy of the Aran-Duchenne type of syphilitic origin.

Merle's[6] case was one in which the only important symptom was muscular atrophy of all four limbs progressing during a period of 15 years. At necropsy many cells of the anterior horns were wanting, a slight thickening of the vessels and small collections of round cells in the pia were found; evidently it was a syphilitic process.

Vix[7] refers to a case of muscular atrophy reported by Oppenheimer. The atrophy began in the upper limbs, and the symptoms were those of progressive spinal muscular atrophy, and the atrophy extended to the lower limbs. Death occurred with bulbar symptoms. Meningitis was found and was most intense on the ventral surface of the spinal cord. The anterior roots were compressed by the thickened pia, and from this resulted the atrophy of the anterior horn cells and of the muscles. This is a different process from the more common form of syphilitic degeneration.

Vix reports a similar case with the clinical picture of progressive spinal muscular atrophy. Meningitis played the chief rôle, and caused degeneration of the anterior roots. The disappearance of anterior horn cells was secondary. Syphilis seems to have been the cause of the meningitis.

Vix refers to some of the literature on the relation of syphilis to progressive spinal muscular atrophy, and adds that the number of published cases carefully studied is too small for a decision as to which process is more common, viz., primary degeneration of the cells of the anterior horns or meningitis with implication of the anterior roots.

S. A. K. Wilson[8] has recently studied the Aran-Duchenne type of muscular atrophy occurring with tabes. He believes that among the types of tabetic muscular atrophy is one which by its

[5] Lannois, Nouvelle Iconographie de la Salpêtrière, Vol. XVIII, p. 593.

[6] Merle, Revue Neurologique, 1909, p. 877.

[7] Vix, Archiv für Psychiatrie, Vol. 47, No. 3, p. 1212.

[8] Wilson, Review of Neurology and Psychiatry, Aug., 1911, p. 401.

progressive nature and its functional distribution is definitely of central origin and analogous to the Aran-Duchenne type. Though not common, it can not be said to be a rarity. While no doubt some cases of this sort, especially some in which true tabetic symptoms are not prominent, are occasioned by a syphilitic meningitis, there are others where the amyotrophy is the result of a chronic process affecting the anterior horn cells more or less directly, *i. e.*, the accompanying vascular, meningeal, or peripheral changes are not sufficient to have produced it. In such cases it seems justifiable to conclude that the syphilitic toxin has been the cause, more particularly since the lesions are widespread, diffuse and irregular.

If this be true of the Aran-Duchenne atrophy occurring with tabes, we must assume it is equally true of this form of atrophy occurring with spinal syphilis, and if the atrophy be due to a chronic process affecting the anterior horn cells the evidence of spinal syphilis may be very slight.

I have therefore gone over my records and reëxamined nine previously reported cases of motor tract degeneration, and one unreported case which will be reported by S. Leopold, to determine in what proportion the most common finding of spinal syphilis, viz., a lymphocytic infiltration in slight or moderate degree, could be found in the pia; and some degree of this has been found in every case, although in some it was so slight as to be questionable. Lymphocytosis of the spinal fluid is valuable as a sign of syphilis though it is not pathognomonic. If the various types of primary degeneration of the central motor system are ever produced by syphilis, we should find at least some evidence of lymphocytic infiltration of the pia, even in the absence of thickening of the vessels. If this infiltration were not present in a certain proportion of the cases one might well doubt the syphilitic etiology. Its presence does not prove syphilis. The importance of lymphocytosis of the cerebrospinal fluid as a sign of syphilis is widely accepted. It is found in other conditions than syphilis, but so is the Wassermann reaction. Lymphocytosis has therefore a certain clinical value.

Nonne,[9] in his address at the 1911 meeting of the Gesellschaft deutscher Nervenärzte, said that from many clinical investigations and from the cases controlled by anatomical examination, we know that lymphocytosis of the cerebrospinal fluid occurs in the true syphilitic and parasyphilitic diseases of the nervous system, whether they are progressive or have been arrested; whether they are in an incipient or advanced stage. The cause of this lymphocytosis is to be found in meningitis. Lymphocytosis of the cerebrospinal fluid occurs in a considerable number of individuals who have been infected with syphilis but have showed no symptoms. The syphilitic infection may have occurred many years previously without spinal cord or brain disease resulting therefrom. Nonne believes, without asserting it as a fact, that only those syphilitic persons develop nervous syphilis who have a lymphocytosis persisting after antisyphilitic treatment. He believes also that the lymphocytosis may be influenced by antisyphilitic treatment.

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[9] Nonne, Deutsche Zeitschrift für Nervenheilkunde, Vol. 43, Nos. 3–6.

The following are cases of tabes or cerebrospinal syphilis with the Aran-Duchenne type of muscular atrophy, progressive spinal muscular atrophy probably of syphilitic origin, and brief summaries of cases of degeneration of the motor tract, previously reported, which have been reëxamined for evidence of lymphocytic infiltration of the pia and thickening of the vessels.

CASE I. TABES WITH TYPICAL ARAN-DUCHENNE MUSCULAR ATROPHY AND DISAPPEARANCE OR DEGENERATION OF THE NERVE CELLS OF THE ANTERIOR HORNS OF THE SPINAL CORD

M. P., laboratory number 577, entered the Philadelphia General Hospital November 4, 1909, and died there November 1, 1911, in the service of Dr. Mills. At the time of entrance he was said to be 75 or 80 years old. He has been deaf and dumb since birth. He had been active until the last 15 or 20 years of his life, but during these years he gradually became bedridden and blind. Previously he had been able to make his wants known by writing. Notes dictated by me January 15, 1909, when the man was in my service, are as follows:

He points to his eyes to indicate that something is wrong with them. So far as can be determined, he has no facial palsy. Reaction to light is entirely absent in each eye. Convergence reaction can not be tested. The left naso-labial fold is not so deep as the right. The masseter muscle contracts well on each side. He has lost most all of his teeth, but has no difficulty in swallowing solid food. So far as can be determined the extraocular muscles are normal, but it is impossible to make him move the eyeballs on command.

The upper limbs are greatly wasted and the atrophy is more intense in the hands and forearms than in the arms. It is symmetrical in the two upper limbs. The thenar, hypothenar, and interosseous muscles have disappeared, and each wrist joint is very flaccid. Involuntary jerkings occur almost constantly in the upper limbs on active or passive movement, but are not present when the limbs are at rest. The upper limbs are very flaccid at all parts, but can be moved at the shoulders and elbows so that the hands are carried about the head in a very incoördinate manner. The biceps and triceps reflexes are entirely lost on each side. Fibrillary tremors are not present in the upper limbs. Pinprick is perceived promptly in both upper limbs and in the trunk. Tactile and thermal sensations can not be tested with certainty.

The lower limbs are much wasted, the feet and legs more so than the thighs. There is marked talipes equino-varus. The patellar reflex and the Achilles tendon reflex are entirely lost on each side. Babinski's sign is not present. Irritation of the sole of either foot produces no movement of the toes, because of the contracture of the muscles. The great toe on each side is contractured as in some cases of Friedreich's ataxia. Pinprick is promptly perceived in the lower limbs. Voluntary power is almost entirely lost. When the lower limbs are pricked with a pin they are drawn away slightly. The power in the lower limbs is much less than in the upper. Involuntary jerkings are seen in the lower limbs on attempted movement.

On October 26, 1911, notes were made that for a week the man had great difficulty in swallowing; he had been having soft diet but even on this he frequently choked, and the obstruction was removed by mechanical means. He howled much and disturbed the other patients. Difficulty in breathing became evident with signs of broncho-pneumonia. The Wassermann reaction tested by Dr. E. Corson White was negative.

The anterior roots of the cervical and lumbar regions were somewhat wasted. Sections from the lumbar region showed diffuse degeneration of the posterior columns, but the posterior roots were very slightly degenerated. The cells of the anterior horns were not so numerous as they should be, and here and there one of these cells presented chromatolysis and peripheral displacement of the nucleus. A very slight round cell infiltration of the pia was found, and there was some thickening of the pial vessels. Marchi sections showed no recent degeneration.

Sections from the eighth cervical and first thoracic segments showed moderate degeneration of the columns of Burdach, but more intense degeneration of the columns of Goll. The cells of the anterior horns at these levels were very scarce, and those preserved were much shrunken. Rarely one was found presenting chromatolysis. Very slight round cell infiltration was present in the pia. Marchi sections showed no recent degeneration.

The optic nerves and chiasm were intensely degenerated.

Some of the vessels of the pia of this part were thickened, and a considerable round cell infiltration of the pia was present in certain parts.

The paracentral lobules showed slight round cell infiltration of the pia, in some parts the pia and its vessels were thickened. Some of the Betz cells were degenerated.

Case 2. Cerebrospinal Syphilis with Typical Aran-Duchenne Muscular Atrophy and Alteration of the Cells of the Anterior Horns of the Spinal Cord

C. F. W., laboratory number 595, a man 48 years of age, was admitted to my service in the Philadelphia General Hospital in January, 1912, and died there February 16, 1912. He acknowledged four or five attacks of gonorrhea but had no knowledge of syphilitic infection. His wife gave birth to two children; one died of marasmus, the other of unknown cause. Two or three years ago he was in the venereal wards of the hospital because of some eruption on the legs.

He had been in poor health nine years, and had been getting gradually weaker. He had had pains in the lower limbs during the past year. He complained on admission of incontinence of urine, weakness of the lower limbs, and atrophy of the muscles of the hands. He had also pain in the shoulders. The atrophy of the hands was noticed a little more than two years before his admission to the hospital. Sexual power had been lost two years.

The right pupil was larger than the left, the irides did not react to light. The hands were greatly wasted, so that the thenar and hypothenar eminences had almost disappeared. The forearms also were much atrophied, and the upper arms less so, and the appearance was that of advanced Aran-Duchenne muscular atrophy. Fibrillary tremors were not observed. The biceps tendon reflexes were slightly exaggerated; the triceps tendon reflexes were normal. Slight ataxia was observed in the finger to nose test. The power in the upper limbs was proportionate to the preservation of the muscles.

The muscles of the thighs and calves were somewhat atrophied. The patellar reflexes were exaggerated. The right Achilles reflex was diminished, the left was normal. Sensation was normal in the lower limbs. The man swayed a little in standing with eyes closed and his gait was a little ataxic. The Wassermann reaction tested by Dr. R. C. Rosenberger was positive.

The man began having convulsions on February 16, at first tonic and later clonic, and had one about every half hour until 3.30 P. M. The left upper limb usually was more implicated in the convulsion than the right, but some attacks were confined to the right side. He died within a few minutes after one of these attacks.

Pronounced round cell infiltration was found throughout the pia over the cord, at the base of the brain, and over the paracentral lobules, also moderate round cell infiltration was found about the vessels within the nervous tissues. The pial vessels were moderately thickened. The pia over the paracentral lobules was thickened. Some of the nerve cells of the anterior horns in the lumbar region presented great alteration, consisting of swelling of the cell body, loss of dendritic processes, chromatolysis, and peripheral displacement of the nucleus, but there was no distinct diminution in the number of the cells. There was a very noticeable diminution of nerve cells in the anterior horns of the eighth cervical and first thoracic segments, especially in the horn of one side, and in both anterior horns the cells preserved presented a shriveled appearance. There was not the prevailing type of degeneration seen in the lumbar region, although a cell here and there presented a similar alteration. The diminution in number and the atrophy of cells was detectable throughout the cervical swelling. The anterior roots appeared to be in good condition.

The crossed pyramidal tracts were much degenerated in the lumbar region, and an area of degeneration was found in the posterior columns on each side of the posterior septum. The posterior columns and the crossed pyramidal tracts were much degenerated in the cervical region. The region of the direct pyramidal tract was much degenerated on each side. The anterior pyramids of the medulla oblongata were not degenerated. The Betz cells of the paracentral lobules were in a good state of preservation.

CASE 3. SPINAL SYPHILIS WITH TYPICAL ARAN-DUCHENNE MUSCULAR ATROPHY

John T., aged 57 years, was a patient in my service at the Philadelphia General Hospital. He was admitted March 14, 1912. His chief complaint on admission was shooting pain throughout his body and limbs, numbness of the legs and arms, and a feeling of compression about the waist.

He had a chancre when about twenty years of age, and "cured" himself with copper sulphate. "Rheumatic" pains have been felt for years, but they have become more severe during the past two years. He has difficulty in controlling the bowels and bladder.

Both upper limbs, especially the left, show atrophy in the forearms and hands of the Aran-Duchenne type (Figs. 1 and 2), *i. e.,* the interosseous muscles and the thenar and hypothenar eminences are much wasted. The hands present marked tremor, so that it is difficult to photograph them. The grasp of each hand is fair. The movements of the upper limbs are free, and the left hand is moved better than the right. Pinprick is not felt in an area two inches in width below the nipples. Touch is perceived here. Biceps and triceps reflexes are very weak.

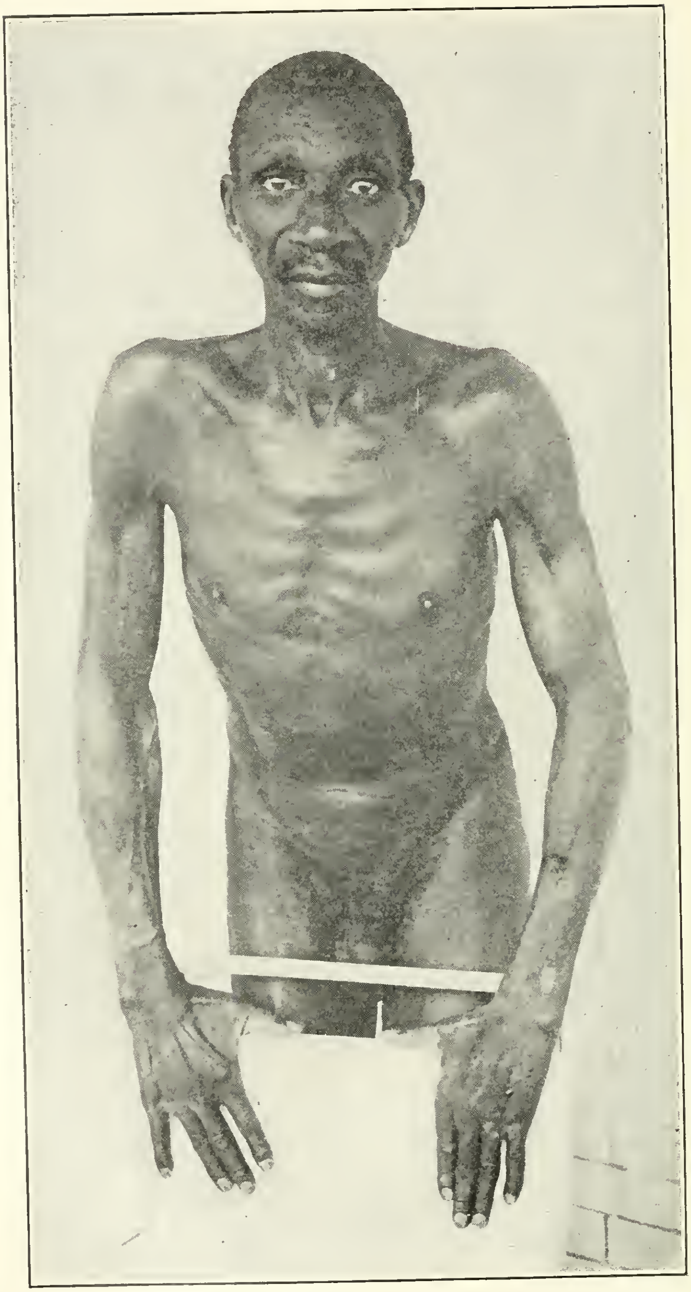

FIG. 1. Case 3. Spinal Syphilis with Typical Aran-Duchenne Muscular Atrophy.

The thigh muscles show loss of power in extension and flexion. The feet are not moved with normal power, especially the right foot. The patellar reflexes are much increased. The lower limbs are spastic. Ankle clonus and Babinski reflex are present on each side at times. No alteration of sensation is found in the lower limbs.

CASE 4. PROGRESSIVE SPINAL MUSCULAR ATROPHY, PROBABLY SYPHILITIC IN ORIGIN

Louis J., aged 61 years, was a patient in my service at the Philadelphia General Hospital in 1912. He acknowledged that he had a soft chancre 35 years ago, and his hair began to come out soon afterward. Seventeen years ago he noticed he was obliged to rest frequently in his work. His upper limbs became tired

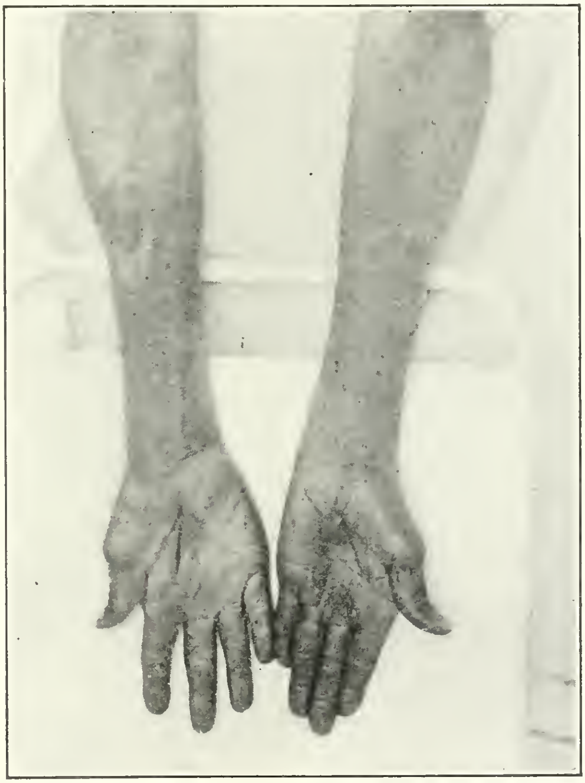

FIG. 2. Case 3. Spinal Syphilis with Typical Aran-Duchenne Muscular Atrophy.

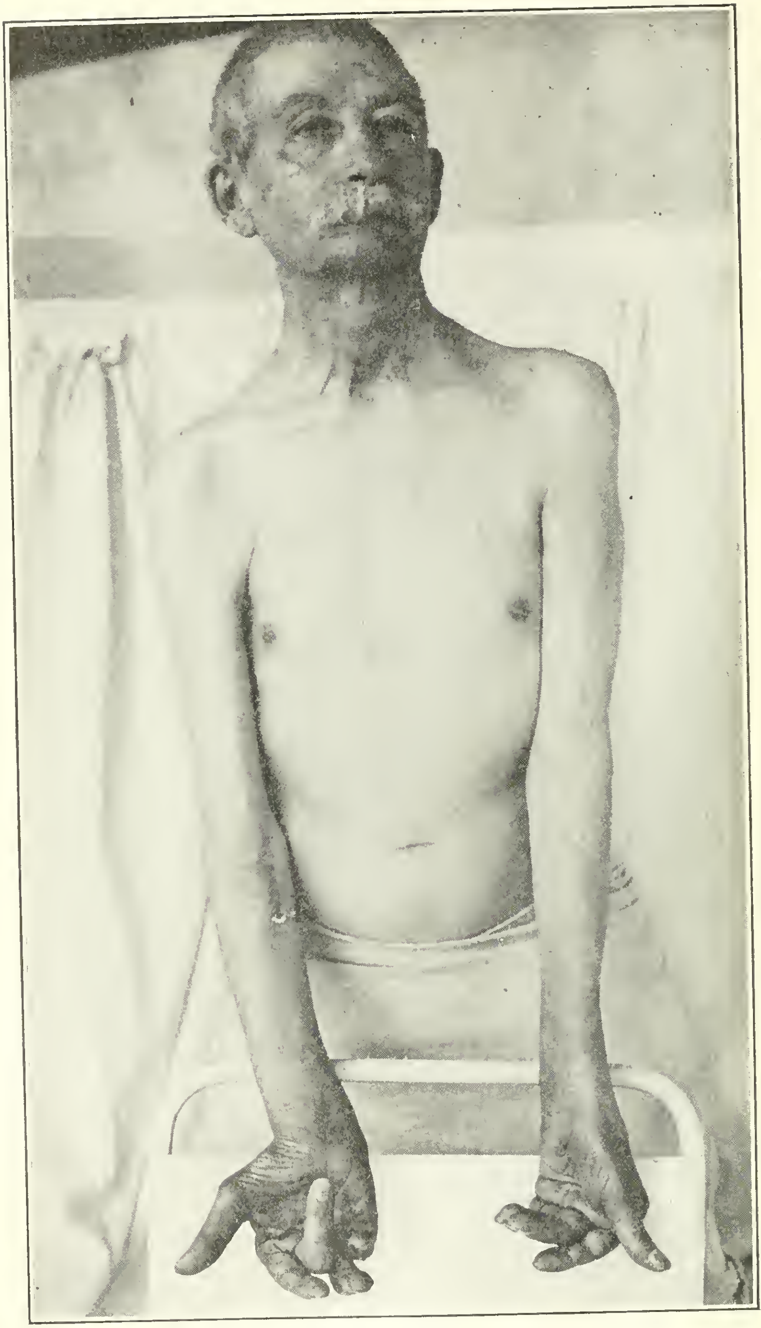

Fig. 3. Case 4. Progressive Spinal Muscular Atrophy Probably Syphilitic in Origin. The Atrophy Developed During a Period of Seventeen Years.

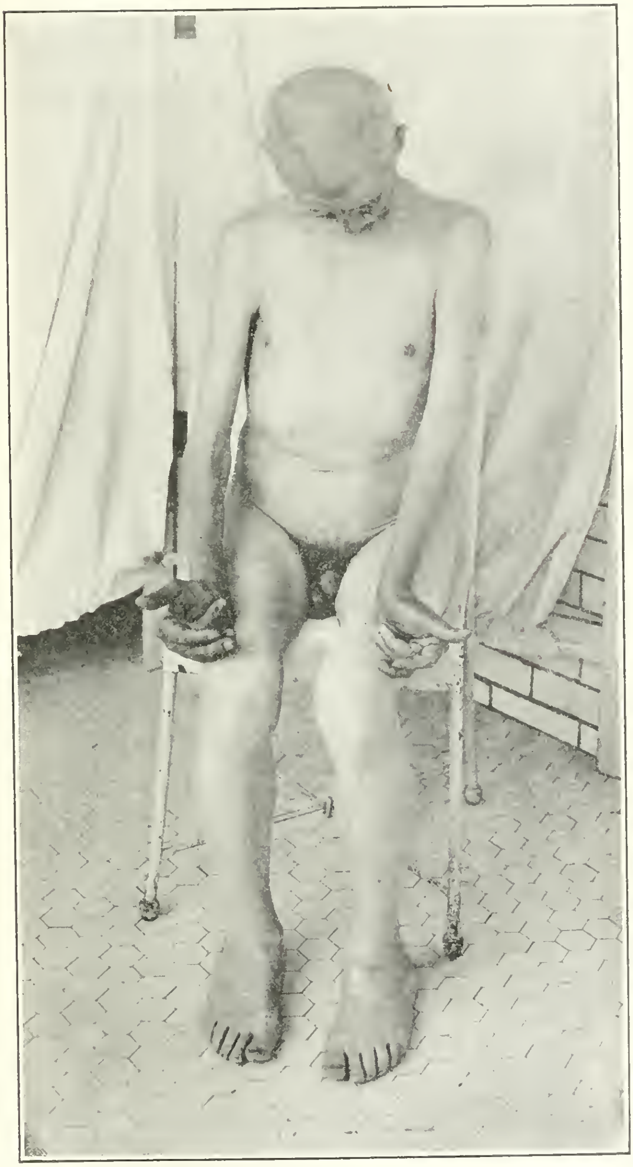

FIG. 4. Case 4. The Man was Unable to Hold the Head Erect, it Fell Either Forward or Backward.

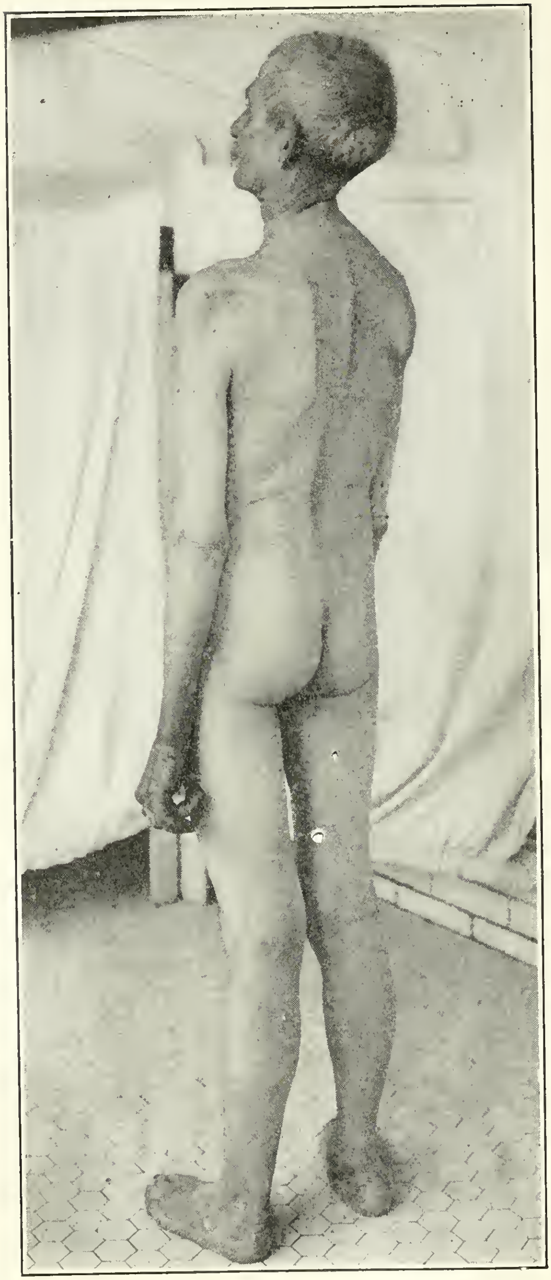

Fig. 5. Case 4. Showing Falling of the Head Backward.

easily, especially the left. He is left-handed. The weakness began in the left shoulder muscles and extended to the left arm, forearm and hand in the order named. About one year later the right shoulder became affected and the weakness then descended the right upper limb. The weakness and atrophy have steadily progressed in the upper limbs. Shortly after the onset of the disease the man had numbness and tingling in the feet and stumbled occasionally when walking. About six years ago the muscles of the neck began to atrophy. He has never lost control of the bowels and bladder. He has had difficulty in swallowing about five years, and this difficulty appeared soon after the atrophy of the neck muscles began. He has had difficulty in chewing a few years.

The man is unable to hold the head erect because of the atrophy of the neck muscles (Figs. 3, 4 and 5); it falls forward on his breast or backward. The muscles of the hands, upper limbs, shoulder girdle and neck are greatly atrophied. Fibrillary tremors are present below the clavicles and in the forearms. Voluntary power in the upper limbs is feeble. He can not abduct the upper limbs to a right angle with the body, or raise them above the head, but he can flex the forearms on the arms and flex and extend the wrists. He can not flex or extend either thumb. The first and ring fingers of the left hand can be extended, the others very little. The grip in each hand is feeble. The biceps tendon reflexes are diminished, the triceps tendon reflexes are a little increased.

The muscles of the thighs and legs are well preserved. The patellar reflexes are slightly exaggerated or normal. Stimulation of the sole of either foot causes a quick upward movement of the big toe. The man staggers considerably when walking with the eyes closed. Touch, pain and temperature sensations in the upper limbs are normal. Dr. E. Corson White reported that the Wassermann reaction with 0.1 c.c. was doubtful; with 0.4 c.c. was positive. Noguchi reaction was weakly positive.

This case presents the appearance of progressive muscular atrophy of spinal origin extending over a period of seventeen years. The venereal sores followed by falling out of the hair, the numbness and tingling in the feet at the onset of the disease, the positive Wassermann when a larger quantity of serum was used, and the weakly positive Noguchi reaction suggest syphilis as the cause of the atrophy.

The following are brief summaries of cases of disease of the motor tract that have been reported by me in connection with others or alone during the past thirteen years, and have been reëxamined for evidence of syphilis.

A CASE TYPICAL CLINICALLY AND PATHOLOGICALLY OF AMYOTROPHIC LATERAL SCLEROSIS.[10]

The patient was a male, aged fifty-three years. For five or six months before admission to the hospital he had complained of pains in both lower limbs below the knees. These pains were thought to be from fatigue. On admission the lower limbs were somewhat spastic and the reflexes were exaggerated. Three years later the weakness and rigidity of the lower limbs had become intense. About this time speech became bulbar in character. The upper limbs gradually became spastic. He died about six years after the beginning of symptoms, after his limbs had become weak, spastic and atrophied, the tendon reflexes had become exaggerated, and bulbar symptoms had developed.

The pathological findings were typically those of amyotrophic lateral sclerosis. The microscopic study revealed degeneration of the nerve cells of the anterior horns of the cervical and lumbar regions, rarefaction of the anterior horns, degeneration of the crossed and direct pyramidal tracts, in the former extending beyond the area of these tracts, and extending as high as the pons, and a slight sclerosis of the posterior columns in the lower cervical and upper thoracic regions.

Recent reëxamination of the slides for signs of syphilis has shown very slight round cell infiltration of the pia of the anterior septum in the cervical region. It is one of the cases with the least lymphocytic infiltration. The vessels are not thickened.

A CASE TYPICAL CLINICALLY AND PATHOLOGICALLY OF AMYOTROPHIC LATERAL SCLEROSIS, WITH DEGENERATION EXTENDING TO THE MOTOR CORTEX[11]

A man, aged fifty-five years, began to have dysphasia, dribbling of saliva, and loss of power in the upper and lower limbs. These symptoms increased until he became confined to his bed and almost completely paralyzed. The tendon reflexes were exaggerated, and fibrillary tremors were noticed in the tongue and other muscles. Sensation was not disturbed. Muscular atrophy was intense. Death occurred about one year after the onset of the disease.

The findings were typically those of amyotrophic lateral sclerosis. The anterior roots of the spinal cord were atrophied. The nerve cells of the anterior horns were degenerated, especially in the cervical region. Degeneration in the motor tracts extended from the cerebral cortex to the lumbar region of the spinal cord.

Reëxamination of the sections revealed slight accumulations of round cells in the pia of the lumbar region (Fig. 6), more here

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[10] Dercum and Spiller, THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Feb., 1899.

[11] Spiller, Contributions from the William Pepper Laboratory of Clinical Medicine, 1900.

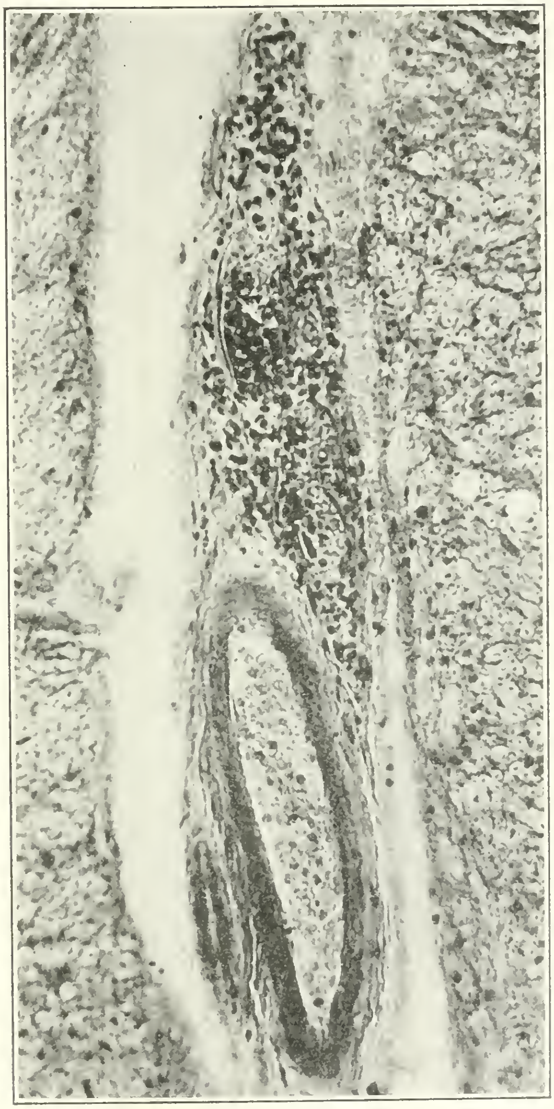

Fig. 6. Amyotrophic Lateral Sclerosis with Degeneration Extending in the Motor Tract to the Motor Cortex. Death in about One Year from the Onset of the Symptoms. Collections of Lymphocytes were Found in Moderate Amount. (Photograph by Dr. A. J. Smith.)

than at higher levels; the vessels were not thickened. This case is especially important on account of the short duration because it is in a case of this kind lymphocytic infiltration would be most likely to occur.

A Case of Primary Degeneration of the Motor System[12]

Sarah L., about sixty-two years of age, became spastic and difficulty in swallowing developed. Gradually all four limbs became paralyzed and control of the bladder and bowels was lost.

The findings were degeneration of the pyramidal tracts and of the nerve cells of the anterior horns and medulla oblongata.

Reëxamination of the sections has shown very slight round cell infiltration of the spinal pia of the anterior septum in the lumbar region.

A Case of Primary Degeneration of the Pyramidal Tracts[13]

Alice G., aged fifty years, developed suddenly weakness in the left upper limb with loss of speech. The power of speech was regained after a day or two, but was never again normal. The weakness almost disappeared in the left upper limb after about three weeks. Two years after this attack she noticed that she was weak in her lower limbs, and the weakness gradually increased, so that walking became difficult. The reflexes in all the limbs were exaggerated, and the Babinski reflex was obtained. No objective sensory disturbances were found and no pains were felt in the limbs. Muscular atrophy did not develop. The reaction of the irides was sluggish.

Degeneration of the pyramidal tracts was found, extending as high as the pons, but not above. The other tracts of the cord were normal. The cells of the anterior horns of the spinal cord were in part diseased.

Reëxamination of the sections from this case revealed a slight round cell infiltration of the pia of the cervical region, but no thickening of vessels.

A Case of Progressively Developing Paralysis, at First of Hemiplegic Type, from Primary Degeneration of the Pyramidal Tracts[14]

Simpson, a man, aged sixty years, developed gradually hemiplegia of the right side, the lower extremity being weaker than the upper. After several years the left lower extremity also became paralyzed, but not to the same extent as the right. The tendon

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[12] Spiller, Robertson and Wadsworth, The University of Pennsylvania Medical Bulletin, June, 1901.

[13] Spiller, Journal of Nervous and Mental Disease, 1902, p. 265.

[14] Mills and Spiller, Journal of Nervous and Mental Disease, 1903, p. 385.

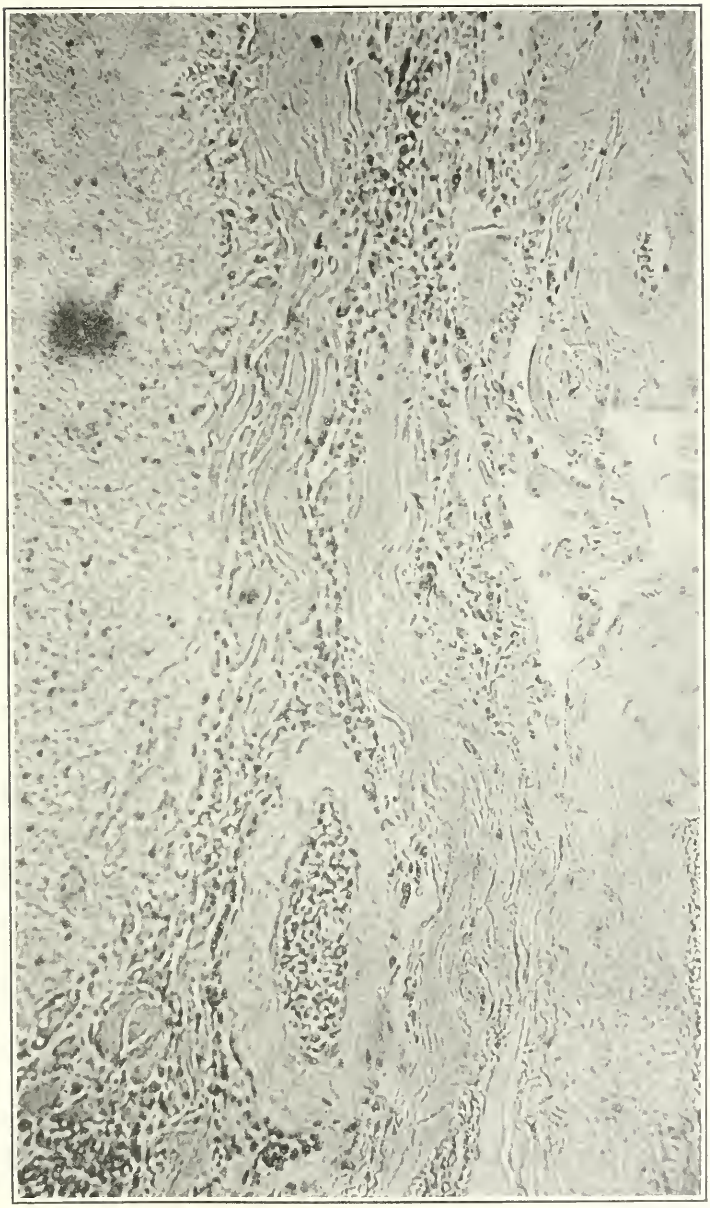

FIG. 7. A Case of Primary Degeneration of the Pyramidal Tracts. Lymphocytic Infiltration of the Pia and Some Thickening of Vessels were Found. (Photograph by Dr. A. J. Smith.)

reflexes were all markedly exaggerated and the Babinski reflex was present. Sensory symptoms were absent.

Microscopical examination showed intense and long-standing degeneration of the right crossed and the left direct pyramidal tracts, the degeneration extending into the pons; comparatively recent degeneration of the left crossed and of the right direct pyramidal tracts, traced by the method of Marchi into the lower part of the right internal capsule. The nerve cells of the anterior horns showed little if any degeneration. The case was one of primary degeneration of the motor tracts. Cellular infiltration of lymphocytic character, of moderate intensity (Fig. 7), was found in the pia about the cerebral peduncles and pons. Some of the vessels were thickened.

A Case with the Symptoms and Findings of Amyotrophic Lateral Sclerosis[15]

Kate D., of middle age, became paralyzed in the right side of the body and face, and within a year also in the left upper and lower limbs, and lost the power to speak. She became greatly emaciated, especially in the muscles of each hand. The thenar eminence was greatly wasted. Fibrillary tremors were present in each upper arm. Spasticity and exaggerated reflexes were found in the upper and lower limbs. The lips were much wasted, and the patient was unable to blow out a lighted match. The tongue was extremely atrophied. The lower limbs were wasted, but not so much as the upper.

The microscopical findings were those of amyotrophic lateral sclerosis, viz., degeneration of the nerve cells of the anterior horns and degeneration of the pyramidal tracts, and of the nerve cells of the hypoglossal nuclei.

Reëxamination of the sections of this case has shown slight round cell infiltration of the spinal pia, especially in the pia of the anterior septum near the base of the septum in the cervical, thoracic and lumbar regions; very little if any implication of the pia about the cervical cord, and no thickening of vessels.

A Case of Spastic Paraplegia from Primary Degeneration of the Pyramidal Tracts[16]

Wm. B., aged forty-two years, had an ulcer near the ankle about four years. Intense spastic paralysis developed slowly in the lower limbs without pain and without atrophy, but contractures and Babinski's sign were present. Sensations for touch and pain were normal in the lower limbs. The upper limbs were almost normal.

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[15] Spiller, University of Pennsylvania Medical Bulletin, Jan. and Feb., 1905.

[16] Spiller, University of Pennsylvania Medical Bulletin, Jan. and Feb., 1905.

The findings were those of primary degeneration of the pyramidal tracts. The nerve cells of the anterior horns were very little if at all altered.

Reëxamination of the slides has shown very slight round cell infiltration of the spinal pia in the cervical and lumbar regions, and no thickening of vessels.

A Case of Progressively Developing Paralysis from Primary Degeneration of the Motor System[17]

Julia S., a woman forty-eight years of age, gradually became paralyzed in the right lower limb, and after a year in the right upper limb. Then the left lower limb became weak, and speech became affected. She had no pain. The paralysis of all the limbs was spastic in type and the reflexes were exaggerated. The muscles of the right hand were atrophied.

The findings were degeneration of the crossed pyramidal tracts, especially of the left, and some degeneration of the cells of the anterior horns.

Reëxamination has shown slight round cell infiltration of the pia in the cervical and lumbar regions, without thickening of vessels.

A Case of Progressive Spinal Muscular Atrophy[18]

A man, aged thirty-seven years, probably syphilitic, developed suddenly paralysis of the group of muscles innervated by the left peroneal nerve; complete loss of faradic irritability occurred in a week, and this loss was followed by rapid wasting and slight diminution in size of the entire leg; the deep reflexes were preserved and even increased, but not excessively. The symptoms were confined to the left lower extremity and were without change during three months; no fibrillary tremors, sensory disturbance, weakness of the hands, nor cranial nerve symptoms developed at this time. One year later there were slight general emaciation, complete wasting of the thenar and hypothenar eminences, and, to a less extent, of the interosseous muscles; atrophy and paresis of the tongue and pharynx, fibrillation throughout the body, including the tongue, and still moderate exaggeration of tendon reflexes. There were no marked electrical changes, except in extremely wasted muscles, and no bladder, bowel, nor sensory disturbances.

The important lesions were disappearance of many of the nerve cells of the anterior horns of the spinal cord and of cells of the motor cranial nerves in the medulla oblongata, degeneration of the anterior roots of the lumbar and sacral regions (cervical region could not be determined), and of the motor nerves of the medulla oblongata, and numerous small hemorrhages in the gray matter, especially in that of the spinal cord, and in the spinal pia. A distinct but not intense round cell infiltration was found within the pia, even in regions where there were no blood corpuscles. The blood vessels were slightly thickened.

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[17] Spiller, University of Pennsylvania Medical Bulletin, Jan. and Feb., 1905.

[18] Moleen and Spiller, American Journal of the Medical Sciences, Dec., 1905.

[Reprinted from The Journal of Nervous and Mental Disease, Vol. 39, No. 9, September, 1912.]

A CASE OF PROGRESSIVE MUSCULAR ATROPHY WITH NECROPSY, PROBABLY SYPHILITIC IN ORIGIN

By S. Leopold[1]

PHILADELPHIA

Confusion has existed in the exact classification of the muscular atrophies ever since Aran and Duchenne first called attention to them under the syndrome of progressive muscular atrophy; and even today this confusion exists and has been increased by the addition of such types as the amyotonia congenita and the Werdnig-Hoffmann form.

An excellent discussion of this subject may be found in Dr. Spiller's recent article in Osler's System, so that it is needless to dwell upon it here. Suffice it to say the sharp distinction between the various forms has been modified. Chronic anterior poliomyelitis and progressive spinal muscular atrophy are used synonymously by many. The question of the distribution of the atrophy in these two diseases, or the fact that the atrophy precedes or follows the paralysis, has little import.

For the most part the constant pathological feature in these two types has been the disappearance or atrophy of the anterior horn cells. The changes in the white matter have been considered secondary. Degenerations have been noted in the anterior roots and in the muscles. The study of the meninges and blood vessels has yielded little of value. The etiology is still uncertain.

The case that I report is important in that it represents a transitional stage between the progressive muscular atrophy and amyotrophic lateral sclerosis, and also because it presents evidence of syphilis. Clinically, it showed the picture of progressive spinal muscular atrophy, while pathologically the lesions were those of amyotrophic lateral sclerosis in a very mild form.

H. R., aged 50, a baker by occupation, noticed in 1906 that he had some difficulty in opening the lid of his watch. This weakness of the fingers and thumb began in the left hand and one and one half years later involved the right hand.

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[1] From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania. I wish to thank Dr. Spiller for the privilege of reporting this case and for his kind aid and criticism.

Family History.—He thinks his mother had the same disease. One brother and two sisters are living and well. His wife has four children. She had on miscarriage four years ago which was self-induced.

Social History.—The patient smoked excessively; had never used alcohol to excess. He denies all venereal infection. No history of trauma is obtained. He had typhoid fever nineteen years ago. He had blood-poisoning two years before onset of his present trouble. Duration of his present condition has been three years. At the present time, February 1, 1909, he complains of weakness and inability to use the arms and hands, with some weakness in his left leg. He has no pain in any portion of his body. He does not suffer from bladder or rectal trouble. Heart, lungs and other viscera apparently are normal.

Neurological Examination.—The pupils are equal; they react to light and in accommodation. There is no disturbance of the ocular muscles. The man wrinkles the forehead equally well on both sides and draws up the corners of the mouth on both sides. The tongue protrudes in the median line; fibrillary tremors are present. Speech is somewhat thick. The patient states that at times he almost chokes while eating, and that when he yawns a cramp is felt in the throat. Sensations to pain, temperature and touch are normal in the face.

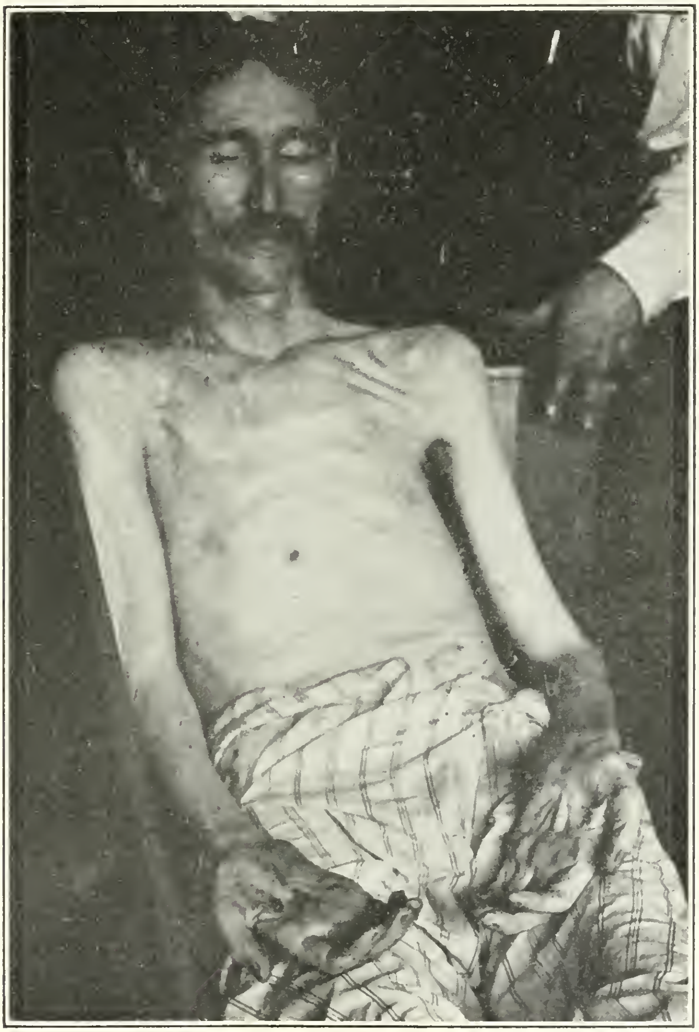
Examination of the chest shows wasting of the muscles anteriorly and posteriorly, and marked fibrillary tremors are seen in all the muscles. The hands, arms and shoulder girdle show a similar wasting of the muscles. Fibrillary tremors are also present. The biceps tendon reflex is absent on both sides. The triceps tendon reflex is present on both sides. Tactile, pain and temperature sensations are preserved on both sides. The grasp is weak in each hand; he can elevate each arm slowly but fairly well. The thenar and hypothenar eminences have disappeared in each hand, and the well recognized conformity of the praying hand of German writers is noted.

The patellar tendon reflexes are not increased; if anything they are somewhat diminished. The Achilles tendon reflex is normal. Babinski's sign is absent on each side. No ankle clonus, and no spasticity are to be noted. Pain, temperature and tactile sensations are preserved in the lower extremities. He has some power in the muscles of the lower extremities.

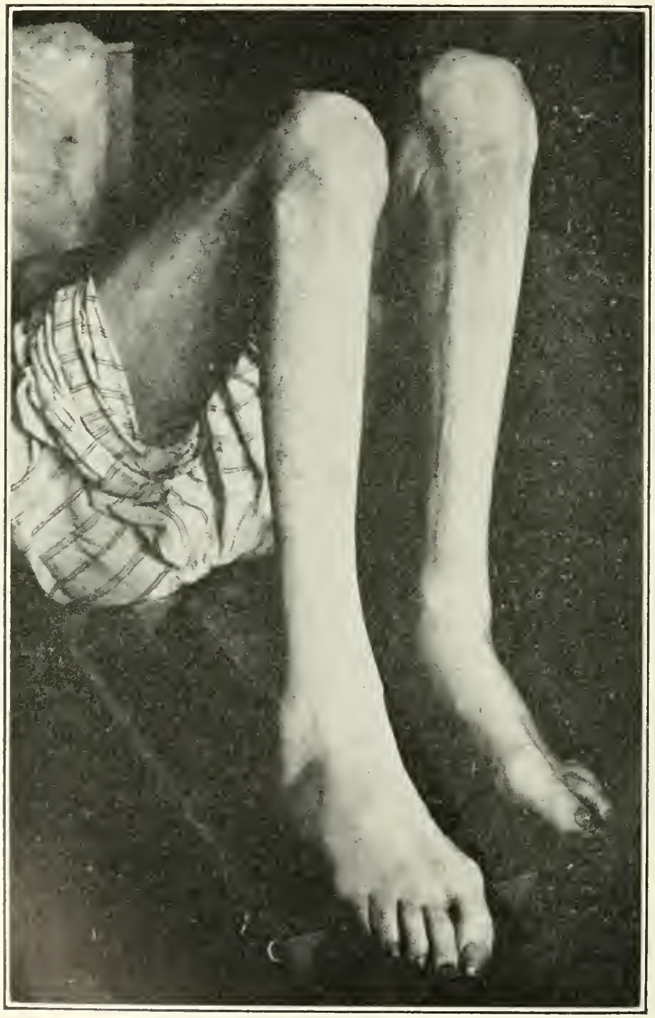
Electrical Examination.—The thenar eminences give no response either to the faradic or the galvanic current. The hypothenar eminences give some response to both currents, but reactions of degeneration are obtained. The muscles of the forearms, arms and shoulders show response to both currents, normally quick and unchanged as to quantity or quality.

The following notes were taken during several examinations covering a period of three months until his death in May, 1910:

The man has no disturbances of sensation in any portion of the



body. The atrophy of the tongue has become more marked. The speech is bulbar in type. The atrophy of the hands and forearms has progressed, and is more marked on the right side. The arms and shoulders show atrophy but to a less degree than do the hands and forearms. He can flex the fingers of each hand, but cannot extend them. The supinators are still active on each side. The atrophy in both legs is considerable. He cannot raise the foot



when the heel is kept on the ground. The gait is steppage. The tendon reflexes in the upper extremities are lost. In the lower extremities the Achilles tendon reflex is still normal, and the patellar tendon reflex is obtainable on both sides. No spasticity is present in either extremity. Ankle clonus and the Babinski sign are absent on each side.

Patient has become quite emotional, alternately laughing and crying at times. During the last month had several severe choking attacks, with paroxysms of coughing.

On the afternoon of May 6, 1910, a decided change took place in his condition, the pulse rate increased to 120, the respiration to 36. There were marked dyspnea and orthopnea. The patient was conscious until three hours before his death, when he lapsed into a stupor, with Cheyne-Stokes respiration.

Necropsy was performed two hours after death; only the brain and spinal cord were removed. No gross changes were noted either in the membranes or on the surface of the brain.

Summary.—Male, aged 50, with doubtful heredity, but with a history of exposure to extreme heat, developed a gradual weakness and wasting in the muscles of the left hand and arm. One and one half years later the other extremity became involved, and a year later the lower extremities became affected. The disease extended over four years. Bulbar symptoms developed during the last six months. Besides the atrophy there were noted fibrillary tremors, reactions of degeneration, preservation of all forms of sensation; absence of spasticity, ankle clonus or Babinski's sign, and no sphincter disturbances. Emotional disturbance developed in the latter part of the disease.

Patellar tendon reflexes were preserved but not increased. The biceps tendon reflexes were absent but the triceps tendon reflexes were preserved on both sides.

Microscopical Examination.—Sections were taken from the lumbar, thoracic and cervical segments of the cord, from the medulla oblongata, pons and paracentral regions. They were stained by the methods of Weigert, Marchi, Nissl and by hemalum and eosin. Some sections of the cord at the various levels were stained by the Bielchowsky method.

Marchi and Weigert Hematoxylin Methods.—Distinct degeneration and sclerosis are noted in the lateral columns in all the levels of the cord, and are especially marked in the lumbar and cervical regions. Both sides are about equally affected and with the Weigert stain the alteration can be detected with the unaided eye. While not so intense as in some cases of amyotrophic lateral sclerosis, it is sufficiently marked to present the appearance of a lateral sclerosis. This sclerosis is systemic in character. The anterior roots show a similar sclerosis.

Nissl Stain.—The various levels show intense involvement of the ganglion cells in the anterior horns. There is marked reduction in number; in some of the sections only three or four cells are seen. Many of the remaining cells show intense chromatolysis and pigmentation and are considerably atrophied. In many cells the form is distorted and the nucleus is displaced or has disappeared. In the cervical levels the picture is more intense. In the medulla oblongata the ganglion cells of the hypoglossal nucleus show a similar picture.

Hemalum and Eosin.—In the various levels the sections show a very slight thickening of the meninges, not sufficient to be considered pathological. The same mild sclerosis is seen in the walls of the vessels. The bloodvessels are congested throughout, especially in the cervical level. There is some perivascular edema, and around some of the vessels a moderate round cell infiltration is noted. It is probably indicative of syphilis. Glial proliferation is noted in the crossed pyramidal areas.

The paracentral lobules, medulla oblongata, pons, and optic chiasm show no changes in their meninges. The bloodvessels show the usual amount of congestion. The Betz cells show some atrophy and degeneration.

A summary of the microscopical findings is a marked atrophy and loss of the ganglion cells in the anterior horns, with moderate distinct sclerosis of the crossed pyramidal tracts; moderate sclerosis of the vessels of the spinal pia with moderate lymphocytic perivascular infiltration.

While the patellar tendon reflex in this case was distinct, it was never exaggerated and at no time could spasticity or the Babinski sign be elicited. Several observers have reported cases with the preservation or even slight increase of the tendon reflexes, and the question naturally has arisen are these cases of amyotrophic lateral sclerosis. Oppenheim does not regard exaggeration of the tendon reflexes as sufficient evidence to make a case of progressive muscular atrophy one of amyotrophic lateral sclerosis, because a slight increase could be due to neurasthenia, while Dana considers it unnecessary confusion of symptomatology to transfer the diagnosis even if there arise a little spasticity; he limits the term amyotrophic lateral sclerosis to those cases which only from the beginning and dominantly show the spastic and contracting type of muscular atrophy. While most observers today consider chronic anterior poliomyelitis and progressive spinal muscular atrophy as synonymous terms, and separate the amyotrophic lateral sclerosis, others (Marie, Gowers) would group them all under the same heading. This difference in opinion may be attributed to the point of view. Some emphasize the clinical and others the pathological features. Those who have emphasized the clinical picture see many varieties. As early as 1888 Nonne separated three varieties of chronic anterior poliomyelitis, and Raymond and Cestan in 1905 gave clinical dignity to four varieties of amyotrophic lateral sclerosis. It is doubtful if such refinement of diagnosis or classification is essential. Considered pathologically, the chief distinction between the two diseases revolves around the implication of the crossed pyramidal tracts. In some cases, as in mine, this characteristic feature loses some of its distinctness. Some portion of the white matter has been found degenerated in nearly all the cases reported, but that portion has usually been the anterolateral ground bundle, and should not be confounded with the degeneration found in the crossed pyramidal tracts. Dejerine reports two cases in which no portion of the white matter showed any change.

In nearly all the cases the anterolateral ground bundle showed degeneration in more or less intensity, viz., in Dubil-Charcot's, Strümpell's, Oppenheim's, Moleen and Spiller's, Cassirer and Maas, etc.

The changes in the crossed pyramidal tract were noted by the Marchi method in some of the cases, viz., those of Bielchowsky, Moleen and Spiller, Aoyama, Grunow, and others.

" It is questionable," says Spiller, " whether these cases should be classified as progressive muscular atrophy or amyotrophic lateral sclerosis." He would rather consider them as progressive muscular atrophy, but as marking a transitional stage to amyotrophic lateral sclerosis.

Few cases show the sclerosis of the crossed pyramidal tract so distinctly as does the case reported, so that with the Weigert stain it may be recognized with the unaided eye. In Hammond's case the posterior columns were normal, the direct and crossed pyramidal tracts and Gowers' tract were degenerated. Several observers consider the case as one of combined sclerosis.

Dreschfield's case also showed marked atrophy of the crossed pyramidal fibers, but mostly in the cervical region. In Pal's case a distinct history of syphilis was noted. Grunow's case showed isolated sclerotic areas in the crossed pyramidal tracts.

Sainton reports a case as follows: The patient aged 32, with a history of syphilis; the atrophy commenced in the upper extremities, finally involving the legs and feet. At necropsy the columns of Goll and Burdach showed marked sclerosis. The anterior horn cells were degenerated and there was slight sclerosis of the direct and crossed pyramidal tracts.

Raymond and Riklin in 1900 reported several transitional forms. Other cases are on record in which degeneration has been noted in the Goll and Burdach columns, and in the direct pyramidal tract.

Of unusual interest is the case reported by Vix. The onset was sudden with cramp-like seizures in the left arm and loss of power in the fourth and fifth fingers. Two years later atrophy was noted in that hand. The right hand became involved in the same manner the following year. Ten years after the patient presented the complete clinical picture of a progressive muscular atrophy.

The pathological changes showed a chronic fibrous thickening of the pia with round-cell infiltration, especially in the anterior portion of the cord, and most intense in the cervical region. The pial septa and larger vessels of the cord and medulla oblongata showed round-cell infiltration. This chronic meningitis caught the anterior spinal roots and the anterior horn cells were atrophied. The white matter was degenerated in the posterior columns and along the entire periphery. Fresh degeneration by Marchi was noted in one of the crossed pyramidal tracts in the cervical region. The case was one of syphilitic meningo-myelitis. He quotes a case of Oppenheimer in which the clinical and pathological pictures were similar to those in his case.

It is probable from the pathological changes in my case that syphilis existed. The pia and bloodvessels were slightly thickened, and some of the bloodvessels showed a slight round-cell infiltration. A round-cell infiltration also was found in the spinal pia.

Raymond as early as 1893 pointed out the relation of syphilis to amyotrophic lateral sclerosis, and recently Hoffmann, Leri, and Dana have emphasized this. Leri collected 30 cases of progressive muscular atrophy in which syphilis was the cause; Dana, in 72 cases, found 25 per cent. gave a history of syphilis. Lannois reports a clinical case of progressive muscular atrophy which showed marked improvement under treatment. Leri cites a case of amyotrophic lateral sclerosis cured by antisyphilitic treatment. Other cases have been reported by Van Gehuchten, Dana, Merle, Sainton, Pal, and Williamson.

In the records[2] given to me of 13 cases of progressive spinal muscular atrophy and amyotrophic lateral sclerosis in which the Wassermann reaction was made, two gave a positive reaction.

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[2] I wish to thank Dr. E. Corson White for the courtesy in placing these records at my disposal.

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Aphasia and Agraphia in Some of Their Practical Surgical Relations

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APHASIA AND AGRAPHIA IN SOME OF THEIR PRACTICAL SURGICAL RELATIONS*

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It is no longer sufficient, when the surgeon is directed by the neurologist to cut here or there, for him to find a tumor, abscess or other lesion lurking in the outskirts of his field of operation, or showing a portion of its disturbing presence beneath the edges of the opening. Cerebral localization and focal diagnosis have been so long with us that the surgeon should find the lesion located by his neurologic confrère occupying the central portion of the area exposed.

Aphasia and agraphia are not infrequently the focal symptoms which dominate the clinical picture in a case in which the question of operation is under discussion. While disease destroying any portion of the zone of language may give aphasic and agraphic phenomena, these differ so much in character, degree and in their symptomatic associations that it is possible to separate the calvarium into at least four or five preferred regions for osteoplastic operations. With four of these our paper is chiefly concerned.

MOTOR APHASIA AND MOTOR AGRAPHIA AND THE POSTERIOR EXTREMITIES OF THE THIRD AND SECOND FRONTAL CONVOLUTIONS

Most Important Diagnostic Phenomena.—Motor aphasia and motor agraphia, usually partial, anomia or paranomia, inability to copy or write from dictation when

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* Read in the Section on Surgery of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

both convolutions are involved; ability to write from copy or dictation preserved when the third frontal convolution alone is involved.

Symptom Sometimes Present.—Partial word-deafness.

Symptoms from Invasion of Neighboring Parts.—Jacksonian epilepsy, usually facial or faciobrachial, inability perfectly to maintain attention, hemiparesis.

The stimulus to present this subject of aphasia in some of its relations to surgical procedure was given by a recent case in which operation was successfully performed, in which the clinical phenomena distinctly indicated implication of the cortex of the hinder portion of the third and second frontal convolutions. Similar cases have been reported by McConnell,[1] Mills,[2] Frazier,[3] and others, but additional reports are desirable especially in the light of the differences of opinion which have arisen since the pronouncement of Marie against the third frontal convolution playing any part in speech disturbances. This we do not believe and our conviction is based on personal experience.

An operable tumor in this midfrontal, aphaso-agraphic region is often an endothelioma and occasionally an inert gumma growing from the membranes, especially from the inner surface of the dura, and invading, compressing and nesting itself in the cerebral substance. Such a tumor usually involves both the third and second frontal convolutions in their hinder parts. It is rarely limited to one or the other of these convolutions, although this is possible and has been recorded, especially for the second frontal convolution.

The disturbances of speech and writing are very partial at first, but gradually become more pronounced. The aphasia and agraphia are rarely complete, and the same is true of the associated and concomitant phenomena such as paralexia.

The hinder extremity of the third frontal convolution, according to our view, is only a portion of the cortical motor center for speech, the remainder of this center being the cortex of the preinsula.

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[1] McConnell, J. W.: Univ. of Penn. Med. Bull., July and August, 1905.

[2] Mills, C. K.: Philadelphia Med. Jour., April 20, 1901.

[3] Frazier, Charles H.: Monograph on Tumors of the Cerebrum by Mills, Spiller, Frazier and others (1906).

A patient suffering from a more or less destructive lesion of the region we are considering presents few phenomena of sensory or conceptual aphasia; he understands what is said to him and is able to read silently either ordinary print or script. He is not word-deaf, or only to a very moderate degree. He is not word-blind, letter-blind, number-blind or mind-blind, and has no cortical color disturbances.

A tumor situated and at first restricted to the hinder part of the third or second frontal convolution, or jointly to both of these, may invade the cerebral substance in a direction either caudad or cephalad, especially the former. Irritation of the adjoining face and arm areas of the motor zone proper often causes jacksonian epilepsy, this being most frequently of the brachial or faciobrachial type, although hemispasm and even general convulsions may occur, the initial symptoms in such cases usually being jacksonian. The spasm is of course of the side opposite the lesion.

Tumors involving the third and second frontal convolutions and progressively advancing sometimes give rise to a slowly deepening hemiparesis. Such growths are easily diagnosticated from tumor, hemorrhage or thrombotic softening deeply implicating the lenticular zone (the lenticula, internal capsule, external capsule, claustrum, etc.). In these deep lesions of the lenticular zone both the motor aphasia and the hemiplegia are practically complete and are of a well-known and often described type, the aphasics usually having but a single recurring utterance or one or two such utterances. Agraphia and forms of sensorial aphasia are also important features of the symptom-complex.

The following two cases, in one of which the patient was cured by operation and in the other by the intravenous administration of salvarsan, are illustrative of the types of motor aphasic and agraphic cases most amenable to surgical or salvarsan treatment.

Case 1.—*History.*—W. B., aged 37, referred to Dr. Mills by Dr. U. G. Gifford of Avondale, Pa., was admitted to the University Hospital, Dec. 9, 1911. He denied venereal disease. The patient had suffered at intervals since childhood with severe headaches. During the six years preceding his admission to the hospital these headaches had become almost continuous. During the last two years he had had epileptiform attacks every two or three months, having had seven of these seizures during the month preceding his admission. The attacks, as described by those who had witnessed them, began with the drawing up of the right arm and both legs. In them he had both tonic and clonic spasms. Consciousness was lost and biting of the tongue occurred. He lost control of his anal and vesical sphincters during an attack. His speech had been failing for about one year and had gradually grown worse.

Examination.—The patient could wrinkle his forehead. The right side of the face showed a slight paresis for volitional movements and in emotional expression. Sensation was not impaired in the face. The lips were not cyanosed. The tongue protruded slightly to the right, with a fine tremor. The abdominal and epigastric reflexes were normal, and there was no impairment of sensations of thorax or abdomen. Examination of the upper extremities showed no atrophy, no ataxia on finger-to-nose test, no asterognosis, no impairment of sensation and no tremors. The biceps and triceps jerks were normal. The grip of the right hand was somewhat weaker than that of the left. Examination of the lower extremities also revealed no atrophy, no loss of power, no ataxia on heel-to-knee test, no Babinski sign, no ankle-clonus and no impairment of sensation. The knee-jerks, gait and station were normal. The lungs, heart, abdomen and abdominal viscera were normal. Urinary analysis showed nothing but a faint trace of albumin. Dec. 9, 1911, Dr. deSchweinitz reported the ophthalmic examination as follows:

Vision: Right eye, 6/15; left eye, 6/22.

Right Eye: Decided choked disk at the temporal edge can still be seen. Surface of swelling, 4 D.; down and in from disk a large hemorrhage on surface of retina, about ½ D.; no macular figure.

Left Eye: Choked disk; 5 D. highest swelling. Retina, ½ D.; some hemorrhages in disk; no macular figure. The form field as taken with the hand was normal.

The pupil movements were normal, with no inequality. There was no failure in rotations and no exterior muscle palsy.

The patient showed decided aphasia, chiefly motor. He was inattentive, although at first he seemed to take notice of what was said to him, later appearing to become easily fatigued. When told to put his knife in the hand of the examiner he placed it in his own pocket. When given a series of letters he pronounced the first few correctly, but then seemed to lose himself or become confused and apparently did not recognize letters or numbers. This may have been due to cerebral or visual fatigue.

The patient was not object-blind, nor was he markedly word-deaf. He appeared sometimes to have difficulty in comprehending what was said to him. He was not word-blind.

When asked to spell "medicine," he spelled it correctly, but pronounced it "merchandise." He wrote his name fairly well, but when asked to write "I want to go home," he wrote, "I go go," and at another time "I want to ho." He seemed confused and threw down his pencil. He could add well, but when asked to subtract and multiply he did not do so correctly.

Dec. 16, 1911, it was noted that the patient had right partial facial paralysis. Voluntary movement, as shown by drawing the corner of the mouth up and to the right, was impossible. Emotional expression as shown in laughing was not entirely lost on the affected side. The tongue protruded toward the right, with fine tremor.

The patient greeted the doctors on making their rounds by saying "good morning," but would not say anything else. He had some apparent difficulty in comprehending what was said to him. It was almost impossible to test for sensation, sense of position, etc., because of his lack of language. Much saliva collected in his mouth, and he did not close his right eye well, but he wrinkled his forehead almost as well on the right as on the left. Ocular movements were normal. He had some weakness in the right arm and hand.

COMMENT ON THE PATIENT'S WRITING

Figure 1: A.—This represents the letters written by the intern in alphabetical series down to and including I, then going to M, then continuing from M, omitting R. When the patient came to I he went on saying J, K, M as if they were present in order. His attention was called to his error by the intern writing J, K, M, above M, N, O. The patient then continued correctly until he came to A which he called U, it being next to T in a regular alphabetical series. Those letters and figures following which have a cross over them were called correctly by the patient. Those included between the short lines were miscalled. The figure 4 in the next to the last line was called E, apparently because it is next to D in the alphabet.

B.—This indicates the efforts of the patient to write the sentence "I want to go home."

C.—These figures represent the success and failure of the patient in his efforts at adding. He first added the figures to the right correctly and then, apparently because of the quickly induced fatigue, failed to add correctly those on the left. Beneath the attempts at addition is an unsuccessful attempt to multiply, which was started right but was not finished.

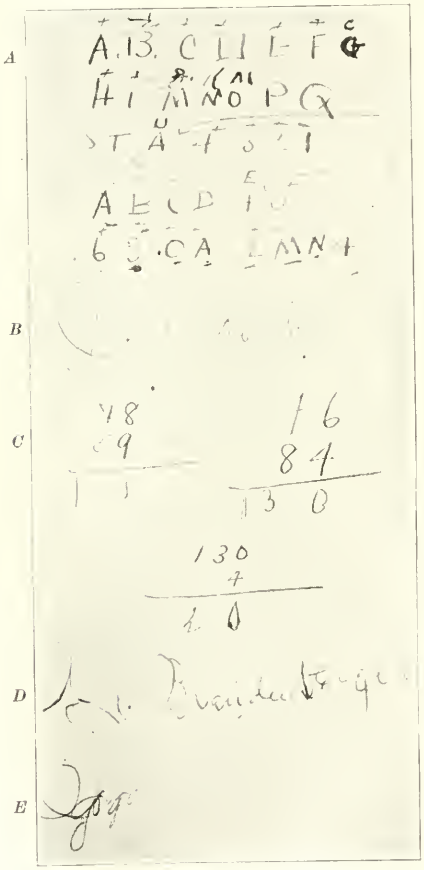

Fig. 1.—Illustrations of the patient's attempts to comprehend and use letters and numbers and his efforts to write.

D.—This is the patient's signature, which is correct but not well written.

E.—This is another effort of the patient to write "I want to go home."

GENERAL REMARKS ON CRANIOMETRIC MEASUREMENTS AND OSTEOPLASTIC OPENINGS

Before describing the operation in this case and before taking up the special osteoplastic openings that are used in aphaso-agraphic cases in general, we shall make a few remarks on craniometric measurements and osteoplastic flaps. In the earlier days of surgical procedure guided by localization, when the trephine openings were comparatively small, it was important to have methods of measurement for the fixation of limited areas and hence arose the various well-known craniometric lines of Reed, Agnew, Anderson-Makins, Horsley and others. These enabled the neurologist and surgeon using well-known data of the relations of various portions of the skull to underlying convolutions to fix with comparative accuracy areas of 1 or 2 inches in diameter. As it is always now regarded as preferable to have openings which in some of their dimensions will be at least 3 or 4 inches, only a few lines need to be determined, but these are of much importance. They are the ectal correlatives

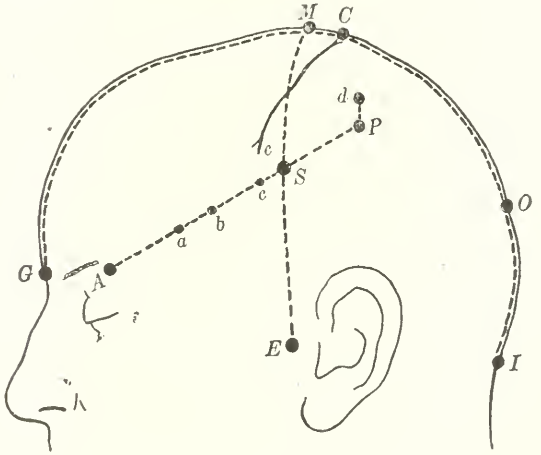

Fig. 2.—Craniocerebral guiding lines traced on a photograph of one of the casts prepared by Professor Cunningham.—(Anderson-Makins). G, glabella; I, inion; M, midsagittal point; A, external angular point; S, squamosal point; P, parietal point; E, pre-auricular point; a, commencement of fissure of Sylvius; b, bifurcation of fissure of Sylvius; d, termination of fissure of Sylvius; Ce, central fissure; O, parieto-occipital fissure; G to I is the sagittal line, A to P the squamosal and E to M the frontal.

of the central fissure, the sylvian fissure and the occipito-parietal notch. With regard to the best openings in aphaso-agraphic cases these markings are all that need to be defined on the skull, with the additional determination of the distance from the midsagittal line for the superior boundary and in some instances the determination of the line which corresponds as closely as possible to the floor of the skull.

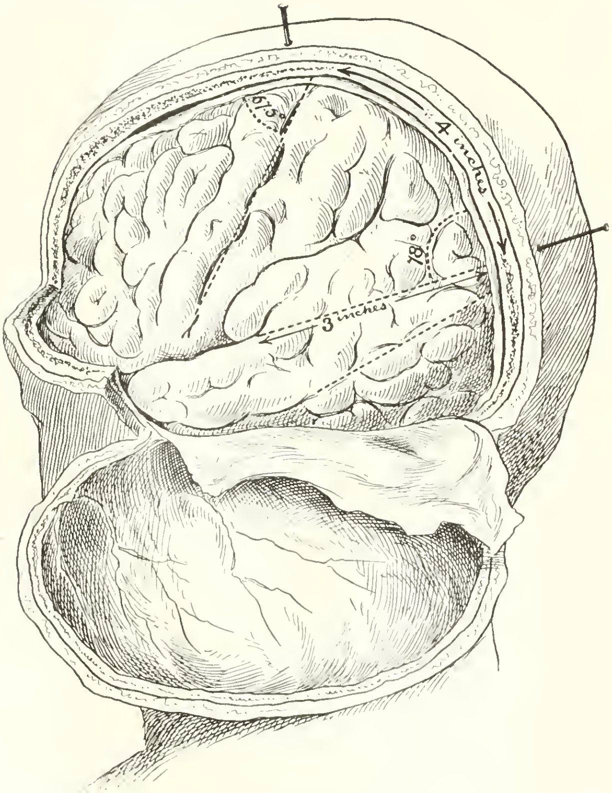

Fig. 3.—Angulation of the central, sylvian and parallel fissures.

In our experience the Anderson-Makins measurements have always been equal to the occasion (Fig. 2).

These it will be remembered are represented by (1) a midsagittal line from the glabella to the inion; (2) a frontal line from the midsagittal point to the depression just in front of the ear at the level of the upper border of the meatus; (3) a so-called squamosal line from the external angular process of the orbit to the junction of the middle and lower thirds of the frontal line and prolonged for about 1½ inches backward. The central fissure is represented by the line drawn from three-fourths of an inch behind the midsagittal point to a point on the squamosal line three-fourths of an inch in front of the junction of the frontal and squamosal lines. The course of the sylvian fissure corresponds closely to the squamosal line. The occipitoparietal notch is usually about 4 inches behind the midsagittal point. In Figure 2 is the well-known representation of these Anderson-Makins lines traced on the photograph of a cast prepared by Professor Cunningham.

ANGULATION OF FISSURES

Neurologists and surgeons during the course of an operation sometimes desire to fix the position of certain fissures as the central, sylvian, and first and second temporal or intraparietal. Mistakes of observation are sometimes made as it is often difficult to do this, especially

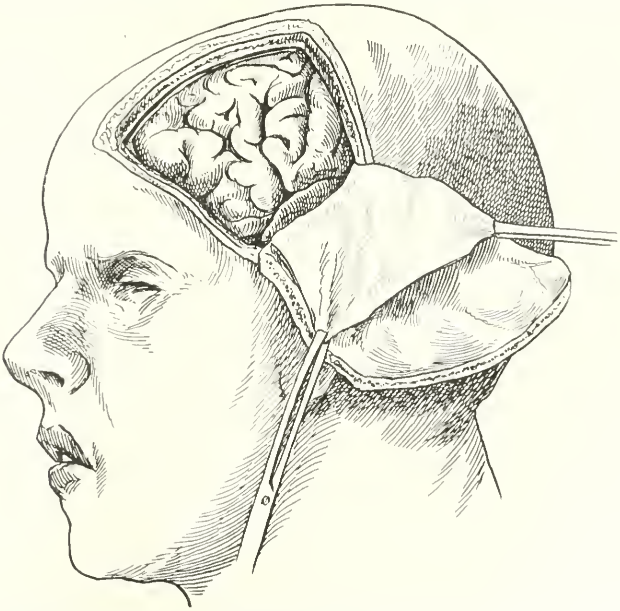

Fig. 4.—Osteoplastic opening for a tumor of the posterior extremities of the third and second frontal convolutions.

with the pia-arachnoid attached to the brain. A retrocentral or precentral fissure may, for example, be confounded with the central or rolandic fissure, or the parallel with the sylvian. A knowledge of the exact angulation of these fissures to the midsagittal line is of assistance in verifying these determinations by visual examination of the field of operation. For this reason we determined on the cadaver and have shown in Figure 3 the angulation of the central, sylvian and parallel fissures.† The angulation of the central fissure to the midsagittal line is of course well known and it is only necessary for us to say that in our dissection, as in those of others, we found that this angulation is not always exactly one of 67 degrees. The angulation of the sylvian fissure is about 78 degrees and the parallel, in its greatest length at least, nearly the same.

OSTEOPLASTIC PROCEDURE IN CASES OF MOTOR APHASIA AND MOTOR AGRAPHIA

Case 1.—*Operation.*—In Figure 4 is shown the opening which was used to uncover the tumor in Case 1. We regard this as

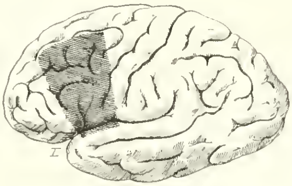

Fig. 5.—The motor-aphasic and agraphic zone (posterior extremities of the third and second frontal convolutions).

the best exposure when a tumor or other operable lesion gives a symptom-complex in which motor aphasia and motor agraphia are the dominating features from partial destruction of the third and second frontal convolutions (Fig. 5). The narrow base line, it will be seen, was just below the sylvian fissure indicated at "s," in Figure 2, and had been made to run parallel with this fissure, rather than with the anteroposterior plane of the brain. The superior line was at about the level of the first frontal fissure or on the skull about an inch or a little more from the midsagittal line and practically parallel with it. The posterior line just cephalad of the central fissure, but not exactly parallel with it, joined the base line just below and behind the lower extremity of the central fissure. The anterior border of the opening ran, of course, so as to cut the second and third frontal convolutions about in half. The measurements of the boundaries of the opening were as follows:

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† A series of osteoplastic cadaveric investigations to identify the relations of cranial areas to the convolutions and fissures beneath them were made by us with the assistance of Dr. Edward M. Williams. Pen sketches were made from photographs of these openings.

Superior boundary	4	inches
Inferior boundary (base line)	1¾	inches
Posterior boundary	4½	inches
Anterior boundary	3	inches

Possibly in some cases a superior line of 3½ inches might be sufficient for the exposure, but as a rule it is better to have the larger opening to provide against contingencies, especially as the prefrontal region is relatively latent and a tumor recognized as located in the posterior extremity of the second and third frontal convolutions by the clinical phenomena might have prefrontal extensions or might be an extension backward from a growth.

After the opening had been made the intracranial pressure was marked; no pulsation was present over the middle portion of the field. When the dura had been removed a small, hard tumor was seen almost exactly in the middle of the opening. After the ligation of two large veins it was found that the growth could be separated from the surrounding brain tissue by the finger and scalpel handle. Great care was taken in enucleating the growth, but this enucleation was completely accomplished.

Microscopic Examination.—The growth was referred to Dr. William G. Spiller of the laboratory of neuropathology for microscopic examination, who reported that it was probably a tuberculoma, although possibly a gumma.

Postoperative History.—After the operation the patient had an uneventful surgical history. The wound promptly healed and the patient had no further attacks except one or two within forty-eight hours of the operation. At first his aphasia and agraphia were quite as marked as before the operation, but his powers of language gradually but steadily improved, so that when he left the hospital in several weeks he was nearly normal in this respect.

MOTOR APHASIA AND AGRAPHIA IN LOCALIZED SYPHILITIC LESIONS AND THEIR TREATMENT BY SALVARSAN AND MERCURIAL INJECTIONS

One matter of much importance for both the neurologist and the surgeon to consider, when the question of operative procedure in a case of focal disease of the brain is under discussion, is whether the lesion which causes the focal symptoms is syphilitic, and if so whether it is amenable to constitutional rather than to surgical treatment, or whether it demands both forms of treatment. This question arises not infrequently when aphasia in some form and degree is prominent. During the time after operation when the patient in Case 1 was still under observation, another case was studied by us which illustrated the point just presented. The patient came to us from a physician in the interior of Pennsylvania distinctly with the idea of having us determine whether medicinal treatment or operation was indicated; if the former, what the method of its administration should be, and if the latter, when the surgical procedure should be undertaken; also, of course, what the locality was of the lesion to be operated on. The patient was carefully examined, and although in some of its features the case was distinctly like the one just put on record in which operation was so successfully performed, we decided that a treatment by salvarsan and hypodermic mercurial medication was more appropriate than operative surgery. Happily the result showed that we were correct in this determination.

The reasons which determined us to favor operation in the first case were in the main the absence of a Wassermann or Noguchi reaction and the persistence of both the aphasic and paretic phenomena. Those which determined us against operation in the second case were the presence of positive Wassermann and Noguchi reactions, the paroxysmal character of the aphasic and agraphic symptoms with the recurrence rather than the continuance of these symptoms, and the absence of any increasing paresis.

Case 2.—*History.*—The patient was a man aged 47, married, who was referred to us in consultation by Dr. C. I. Keen, Dec. 15, 1911. His general health was good until about seven or eight weeks before coming under observation, when he began to be troubled with noises in the left ear. He was treated for this by a laryngologist with the usual local methods—nasal, pharyngeal and eustachian. While traveling in the south two or three weeks before he was first seen, he was taken at midnight with a peculiar seizure. He had, according to the description both by himself and his wife, a typical jacksonian seizure. He awoke with a feeling as if he could not swallow, felt drawing on the right side of his face, this drawing or spasm extending to the arm of the same side; then his tongue began to move or "wriggle," as he expressed it, and he had also some movement of the jaw. He attempted to get up, but in trying to do so fell to the side of the bed. He was soon seen by his wife, who found that he could not speak, although he made efforts to do so. He also exhibited right-sided spasm (jacksonian) when she first saw him. During the same night he had five similar but less severe attacks, those after the first affecting only his speech and face and tongue. His speech was disturbed or apparently impossible for five or ten minutes after each of the spells. The next morning he was all right.

Between the time of these initial seizures and his coming under our care, the patient had several of the milder seizures, in which his tongue and speech were affected and in which he was not completely unconscious. In them he would appear dazed and would attempt to speak, but do so in a mumbling or inarticulate way, apparently conscious of his own difficulties.

It was learned that two years before he was troubled for a time with headache, which passed away, and that again about six months previous to his first seizure the headache had been severe. He referred the pain to the vertex or top of his head, and said it made him feel as if he could not hold up his head. His family physician stated that in the attacks the patient's throat sometimes seemed to be distinctly affected as indicated by some sort of gurgling or distressing sound.

Examination.—An elaborate physical examination was made, this including an investigation of his station, gait, response to active and to passive movements, sensation in all its varieties, astereognosis, diadokokinesis, knee-jerks, ankle-jerks, the Babinski reflex and other reflex phenomena. The results of the examinations were negative, except in two or three respects. These were that the tongue went slightly to the right on protrusion: that there was deafness to a moderate degree in the left ear: that speech was at times blurred, particularly in pronouncing the gutturals; and that the finger-to-nose test showed very slight ataxia or weakness of the right upper extremity.

Course of Disease.—The patient was admitted to the Hospital of the University of Pennsylvania, where he was kept under close observation. During the first week he had a number of the aphasic seizures, usually without facial or limb spasm, although in one or two instances the right side of the face was slightly affected. While he seemed dazed, his consciousness was largely preserved, so that it was possible to investigate his condition as to speech, writing and general mentality. Some of these observations were made both by ourselves and by the resident physicians, Dr. J. C. Birdsall and Dr. T. G. Schnabel.

The patient had an attack on the morning of Dec. 19, 1911. He reported a preliminary numbness in the right side of the tongue. This was followed by convulsive movements of the tongue, which drew it back and to the right. During the attack he could utter sounds, but they were unintelligible. His vision was normal and his eyes were normal to all the usual tests. Smell was preserved. The face at intervals underwent clonic movements, the right side of the mouth being drawn toward the ear. The movements involved mostly the muscles supplied by the lower distribution of the seventh nerve. In the more violent spasms the orbicularis was involved, but not the corrugator supercilii. On some occasions the ear moved quite vigorously. The jaw was not moved. The whole period of the seizures lasted about one hour and twenty-five minutes, interspersed with intervals of a few minutes when the convulsive movements of the face ceased, but the aphasia still continued. The patient could swallow water. Saliva dripped from his mouth at times. No part of his body except the face was involved.

The patient during the period of the attack was agraphic. He held the pen and pencil in a normal way, but could not write. He copied writing when it was placed before him, but with only an approximate degree of accuracy.

Between the convulsive movements the facial paresis was emphasized and the tongue protruded to the right with tremors. The jaw opened normally. So vigorous were the clonic movements of the right face that the left side was pulled over and at times seemed also to twitch. The saliva was forced through the teeth and into the labiodental space, so that a noise was made. The muscles of the floor of the mouth were also involved in the attack.

COMMENT ON THE PATIENT'S WRITING

Figure 6: A.—This represents the efforts of the patient to write from dictation "This is perhaps the longest attack I have had since I have been in Atlanta on Thanksgiving Day."

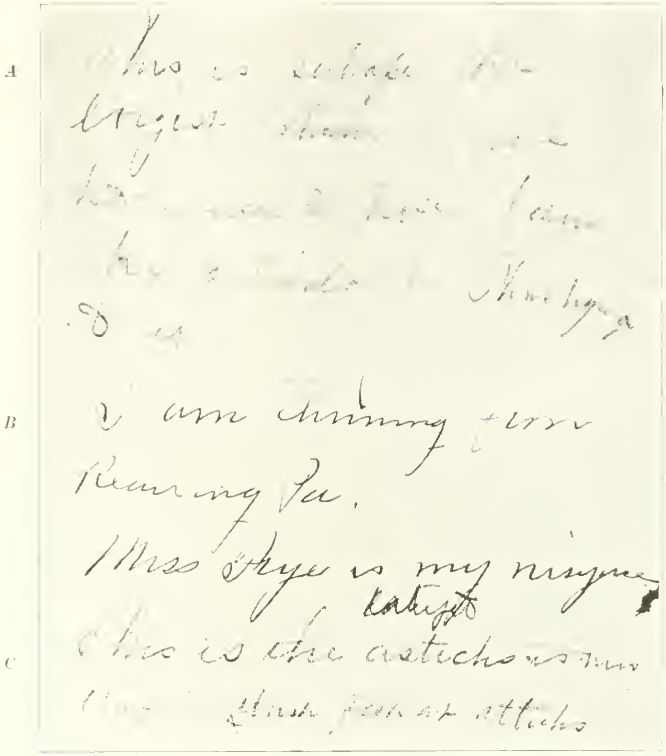
B.—These represent the efforts of the patient to write spontaneously. What he wanted to say was "I am coming from Reading." "Miss Frye is my nurse."

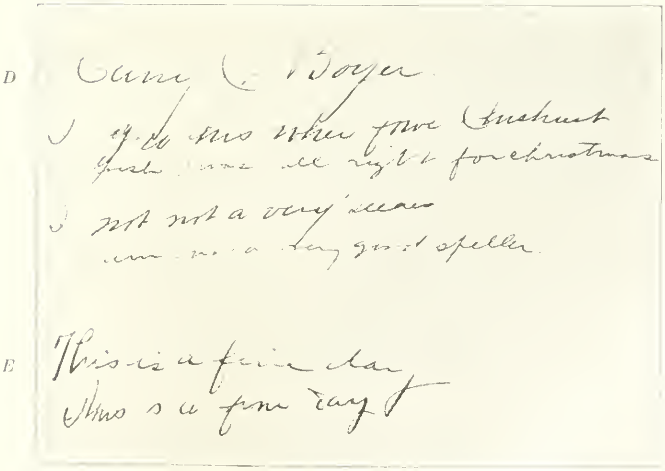
C.—This indicates the effort of the patient to write from dictation "This is the longest attack since I have been in Atlanta."

Figure 7: D.—This indicates the patient's signature; an effort to write spontaneously "I wish I was all right for Christmas"; an excuse which he wrote for his agraphia by trying to say "I am not a very good speller."

E.—This represents writing from copy, the intern having written "This is a fine day."

Dec. 18, 1911. Dr. B. A. Randall reported that there was a faint dulness of the left drum-head, but nothing of moment visible in the ears, nose or throat. "His tuning-fork tests are fair except for reduced bone conduction. There is little suggestion in his eye-grounds of intracranial trouble, present or past. I should recognize only slight or incipient nerve





Figs. 6 and 7.—Illustrations of the patient's attempts at writing during his aphaso-agraphic attacks.

impairment with no localization as between centers, nerve-trunks or terminals."

The following reports of eye examinations were made by Dr. George E. deSchweinitz: "The form and color fields are normal. Each disk is slightly hazy at upper and lower margins. The veins are fuller than normal, but there is no papilledema. The pupils are normal. Vision: Right eye, 4/6; left eye, 4/5."

The blood was submitted to Dr. S. D. Ludlum and Dr. E. P. Corson White for examination and also for an expression of their opinion on the results of the examination, and on salvarsan treatment in some cases which they had observed, after positive determinations by blood-tests. Both the Wassermann and Noguchi methods were used, and the report was that both tests were very positive and indicated an active syphilitic infection.

Drs. Ludlum and White, handing in their report, said: "We have had four cases of positive Wassermann reaction when tumors were present. One was a case referred by Dr. W. G. Spiller, of gumma of the liver with apparently fibroma of the brain. The second was a case of Dr. Kapland, one of visceral syphilis with tumor of the brain. The third and fourth were cases of Dr. Spiller, of hemicraniosis, bone syphilis, with endothelioma underneath. It would seem that this patient's case, although he may have a tumor, is probably syphilitic, and that he should have salvarsan before any operation which might start syphilitic overgrowths, is considered. We do not think that a lumbar puncture is necessary, for the blood is definitely positive. Supposing a lumbar puncture were done and no lymphocytes found, it would indicate a tumor not syphilitic. Knowing definitely that syphilis is present and that the supposed tumor would probably be grown on the syphilitic basis, we think it would be better to give the salvarsan before disturbing the tissues."

Treatment and Course.—Because of this report and the suggestions accompanying it, which were in accordance with our views, it was decided to postpone operation and use salvarsan by intravenous injection. The injections were given by Dr. B. A. Thomas, with all the most approved precautions. Eighteen days after the first injection, as the renewed tests still showed a positive reaction, salvarsan was again given by intravenous injection by Dr. Thomas. About two weeks later the reaction was so weakly positive as to be doubtful. From the time of giving the first injection the patient never had another seizure. After leaving the hospital he reported several times over a period of about three months. The seizures had not recurred and the patient expressed himself as being in absolutely good health.

Since his return home he has been taking 10 grains of sodium iodid and about one-fifth of a grain of protiodid of mercury three times daily, going on with this for three weeks and stopping one week and then continuing again.

Besides the motor aphasic and motor agraphic syndrome which has been discussed and illustrated by the two cases just given, it will be found useful to recognize three other symptom-complexes which may serve the neurologist in guiding the surgeon in the selection of sites for osteoplastic operations. These syndromes are among those which in our experience have been presented in cases of operable tumor, abscess and hemorrhage, especially the former two. They are, in other words, symptom-complexes representing cases which can be successfully diagnosticated and palliated or cured by surgical procedure.

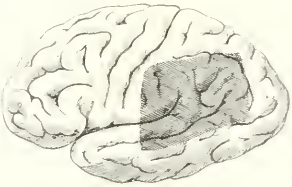


Fig. 8.—Wernicke's zone.

AUDITORY APHASIAS AND WERNICKE'S ZONE

Most Important Diagnostic Symptoms.—Word-deafness, complete or partial, paraphasia, partial motor aphasia.

Symptoms Sometimes Present.—Word-dumbness, paralexia, auditory apraxia, amusia, auditory hallucinations.

Symptoms from Invasion of Neighboring Parts.—Hemianopsia, loss of sense of position and passive movements, astereognosis, impairment of cutaneous sensation, hemiparesis or hemiplegia.

It is sometimes desirable to make in tumor cases an osteoplastic flap which will have for its center the posterior third of the first temporal convolution. This is in cases in which the symptomatology indicates that the growth is centralized and probably has begun here, or, if the case is one of hemorrhage or abscess, that its most destructive force has here been exerted. Such a lesion may be described as one chiefly located in the central portion of the anterior half or two-thirds of Wernicke's zone, which is regarded as including the supramarginal or subparietal convolution, the angular convolution, the first and second temporal convolutions, and the anterior boundary of the middle portion of the occipital lobe as represented in Figure 8. A tumor or other lesion thus localized and centralized will, after its effects are well defined, have for its most important symptoms word-deafness and paraphasia. Partial motor aphasia is often present but may be very partial. Word-dumbness, also usually partial, is often exhibited, and more or less alexia or paralexia because of interruption of the connections between the higher auditory and higher visual centers. The auditory phenomena will, however, be more dominant. Amusia may also be a symptom especially if the growth or its secondary results have advanced in a cephalic direction in the temporal lobe. Hemianopsia comes on in some cases as the growth advances, and if it spread so as to involve the parietal lobe and motor regions, other symptoms may be impairment of cutaneous sensation and especially of the sense of position and passive movement with or without hemiparesis.

In every such case the diagnostician should fix his attention closely on the history of the earliest symptoms shown and the rate and probable direction of the advance of the neoplasm. While some word-deafness will almost invariably be present, this may be very partial at first, becoming more marked as the disease, which in the case of an operable tumor is usually one which develops from the membranes and grows inward so as to displace and destroy cortical and subcortical tissue, extends more and more from its place of origin. As the word-deafness becomes complete, the other diagnostic phenomena, such as paraphasia and the auditovisual symptoms above described, come into more and more prominence. We are speaking, of course, in this connection as in most instances in this paper, of the lesions in the left hemicerebrum in right-handed individuals. In those who are left-handed the same remarks would apply, although perhaps with not quite equal force, to tumors or other lesions similarly situated in the right first temporal and neighboring parts. It is true that cases of destructive lesion of the first left temporal convolution have been recorded which have not given word-deafness, but these are the rare exceptions and need to be accounted for in some special way. The anomia or word-deafness in lesions of this area, while often present, is not so common a phenomenon as in lesions in the middle or more inferior portion of the temporal lobe or in the region of the parietotemporo-occipital junction. Auditory apraxia or mind-deafness, the inability to recognize objects through sound, and auditory hallucinations are present in rare instances. Mind-deafness, however, in most cases seems to require bilateral destruction of the temporal zones, and hallucinations of hearing are inconstant and in the absence of other diagnostic symptoms not reliable for the purpose of diagnosis.

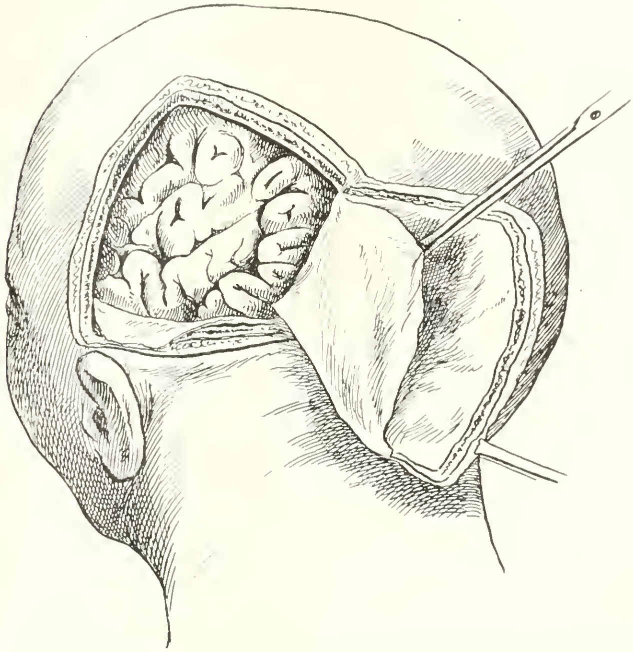


Fig. 9.—Osteoplastic opening to uncover the anterior two-thirds of Wernicke's zone.

In probably one-fourth or even one-third of the cases of destructive lesions involving the auditory portion of Wernicke's zone, word-blindness and alexia are absent, although there may be partial word-blindness and alexia, and frequently paralexia. The power to write spontaneously or from copy may be retained. Writing from dictation will of course be lost if word-deafness is complete or nearly complete.

Retention in whole or, what is more usual, in part of functions like word-hearing, which theoretically should be lost in such cases, is sometimes to be accounted for by the gradual development of the lesion, other neighboring parts taking on the functions, or possibly, as held by some writers, these being acquired by the convolutions on the right side.

OSTEOPLASTIC OPENING TO UNCOVER THE ANTERIOR TWO-THIRDS OF WERNICKE'S ZONE

The opening to be preferred to uncover the anterior two-thirds of Wernicke's zone in the first temporal convolution is that indicated in Figure 9. The lower border of this opening extends from a point slightly posterior to the line from the midsagittal point to the preauricular point at a level of one-half inch above the top line of the zygoma, backward for about 3¾ inches. The anterior border of the opening reaches from the anterior end of the lower limiting line upward for about 4 inches. The superior line is drawn from the upper extremity of the anterior boundary backward for about 3½ inches. The line of breakage is thus formed at the anterior margin of the occipital region, being about 1½ to 2 inches in length. This opening will thoroughly expose the subparietal and posterior halves of the first and second temporal convolutions, as shown in Figure 10. The opening might be made with the line of breakage below or even above, but we believe it will be found most desirable to place it posteriorly as indicated in the figure and description, this from the standpoint both of convenience and of blood-supply to the flap.

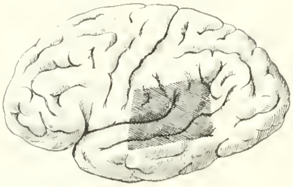


Fig. 10.—Auditory aphasic zone.

ANOMIA (WORD-DEAFNESS) AND THE MIDTEMPORAL REGION (CONCEPT AREA AND NAMING CENTER)

Most Important Diagnostic Phenomena. — Anomia (visual or for all senses), paraphasia, paralexia, paragraphia, partial motor aphasia.

Symptoms Sometimes Present.—Hemianopsia, color aphasia (color dissociation), mind-blindness (visual apraxia), partial word-deafness (auditory apraxia), visual or auditory hallucinations.

Symptoms from Invasion of Neighboring Parts.—Word-deafness, partial word-blindness, letter-blindness, or number-blindness, hemiparesis.

Tumors or other operable lesions are occasionally present in the midtemporal region (third and fourth temporal convolutions), as shown in Figure 11. These may extend in their effects forward to the anterior extremity of the temporal lobe or backward into the occipital lobe, the aphasic phenomena present in such cases forming a symptom-complex which, if the case is studied thoroughly, may be sufficient to define a site for operation. Such an operation for most successful surgical results requires an osteoplastic opening different in position, although adjoining that for the exposure of Wernicke's zone proper. The dominant feature in such a symptom-complex is anomia, or if the lesion is incompletely destructive, paraomia. This syndrome is based on the theory, which is held by us, that in the general zone of speech is situated a concept area intercalated between the sensory percept regions, auditory and visual, and the cortical motor centers for speech and writing, namely, Broca's convolution and the posterior half of the second frontal convolution. Within this area for concrete concepts is a centralized portion which has for its function the registering of the names of objects

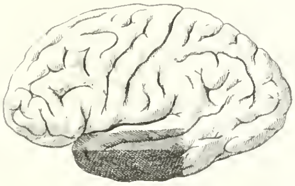


Fig. 11.—Midtemporal aphasic zone (chiefly third and fourth temporal convolutions).

recognized. Cases are reported, for instance by Potts,[4] in which immediately after an acute apoplectic lesion, probably hemorrhage, the patient lost entirely the ability to name objects which he fully recognized either through sight, hearing, touch, taste or smell. It follows, as Potts contended in reporting this case, that the anomia or word-dumbness could be explained only on the theory of the severance of tracts running from all the different percept centers to the motor centers for speech, or from a lesion of the center in which all these tracts become concentrated on their way to the motor or emissive speech areas, or finally from a lesion of the tracts running from such naming center to the motor centers for language. Whether a region of such centralization should be regarded as merely the place of coming together of transcortical or conduction lines, or should be considered as a special center, is a matter of practical indifference in the determination of the position of the lesion for operation. In any case, destruction of such an area or center would result in general anomia, that is, anomia as regards all the senses which might be concerned in concepts related to sensory impressions.

In some instances the anomia has been simply for objects seen, this constituting so-called optic aphasia; in other reported cases it has been for objects touched or handled (tactile aphasia or amnesia); in others for objects seen, touched or handled, etc. When the word-dumbness has been demonstrated by testing the powers of the patient through all the senses, there can be no question as to the possibility and even probability of a particular, more or less strictly localized midtemporal area being the site of the lesion.

The mistake must not be made of supposing that anomia does not occur from lesions differently situated. It may be present as the result of bilateral lesions of the occipital lobe, the lesions of Wernicke's zone, cortical or subcortical, in motor aphasia, and in various aphasic complexes. The word-dumbness is indicative of a somewhat isolated midtemporal lesion only when it presents the peculiar diagnostic symptomatology which has been described in the summary at the beginning of the remarks on this region.

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[4] Potts, C. S.: The Journal A. M. A., May 4, 1901, p. 1239.

Several years ago Mills and McConnell[5] reported a case of tumor (glioma) which had originated in the third temporal convolution, nearly on a line with the posterior branch of the sylvian fissure, and had gradually extended so as to involve in its destruction and surrounding degenerations the fourth temporal convolution, a portion of the second temporal, and the central portion of the occipital lobe. Anomia or word-dumbness was throughout the leading symptom, although the patient had a lateral homonymous hemianopsia, the blind field not including the macula, a temporary partial object blindness, and late in the case, hemiplegia and general sensory motor aphasia. The patient was word-blind but not entirely letter-blind and early had a marked paralexia. She was not word-deaf, and in the early stages not a marked motor aphasic, although largely omitting nouns from her vocabulary. Her anomia or word-dumbness was tactile as well as visual. The case, while somewhat imperfectly reported, had in it elements of positive diagnostic importance and pointed to anomia or word-dumbness for several of the senses as the most important symptom. The symptom-complex was not that of a lesion limited either to the angulo-occipital region or the first temporal convolution, although the case had in its symptomatology some of the symptoms of lesions of both of these aphasic zones. It had most of the features which would enable the neurologist to define a tumor of the midtemporal convolution for the purpose of operation.

This case might have been successfully treated by operation a year or so before death.

Abscesses of the temporal lobe associated with disease of the middle ear not infrequently give the symptom-complex of the midtemporal region, although, of course, symptoms of Wernicke's zone or of the angulo-occipital region may be present when the abscess has notably extended.

Oppenheim,[6] in discussing anomia or word-dumbness under Freund's title of "visual aphasia," says that this visual or optic aphasia is usually associated with alexia, hemianopsia and often with sensory aphasia, but in one case he could discover no trace of word-deafness. He believes that hemianopsia is not a necessary accompaniment, and bilateral foci in the white substance of both occipital lobes may apparently also cause the condition (visual anomia, word-dumbness from sight).

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[5] Mills, C. K., and McConnell, J. W.: Jour. Nerv. and Mental Dis., January, 1895, xxii.

[6] Oppenheim, H.: Text-Book of Nervous Diseases, transl. by Alexander Bruce, 1911.

Oppenheim further says that visual aphasia as well as the auditory-visual form in which the patient does not comprehend from spoken speech what he can perceive by means of the tracts connecting the center for sound-formation with the vision centers, is not an unusual symptom of cerebral abscess from ear disease.

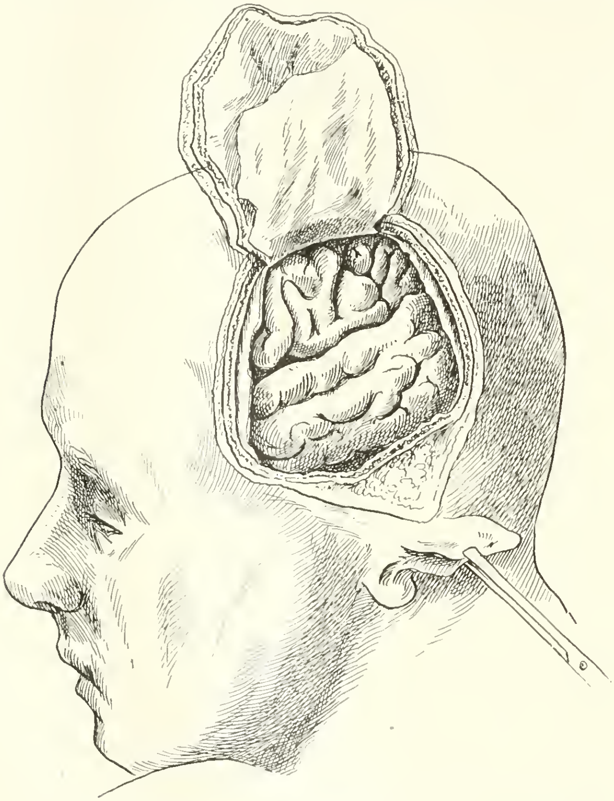

Fig. 12.—Osteoplastic opening to reach the midtemporal conceptaphasic zone.

It will be seen that a lesion of this midtemporal region, especially an abscess, is of much diagnostic importance not only to the neurologist and general surgeon but also to the aural surgeon.

OSTEOPLASTIC OPENING TO REACH THE MIDTEMPORAL REGION

This third opening is made with the inferior line running parallel to the zygoma and about one-half inch above it, extending from 2 fingerbreadths behind the external angular process, backward to a line through the mastoid process. The superior line, parallel to the sylvian fissure, runs forward and backward sufficiently far to intersect perpendiculars from the posterior and anterior ends of the inferior line. The breakage here is made on the superior line and the flap turned upward, as shown in Figure 12.

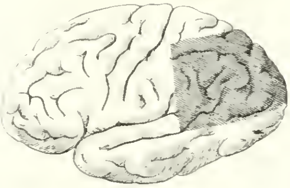

Fig. 13.—The visuo-aphasic angulo-occipital zone.

VISUAL APHASIAS AND THE ANGULO-OCCIPITAL REGION

Most Important Diagnostic Symptoms.—Word-blindness, letter- and number-blindness, alexia, visual agraphia, lateral homonymous anopsia or hemianopsia.

Symptoms Sometimes Present.—Word-dumbness, visual apraxia, color aphasia (color dissociation), visual hallucinations, paraphasia, inability to copy from sight.

Symptoms from Invasion of Neighboring Parts.—Loss of sense of position and passive movements, astereognosis, impairment of cutaneous sensibility, word-deafness, partial or complete, inability to copy from dictation.

When a tumor or other lesion has for its earliest and most centralized phenomena visual aphasias of various forms, the aim of the neurologist and surgeon should be to select an opening which would uncover what is best termed the angulo-occipital region — an area of the brain which includes in its anterior third or fourth the convolutions which curve around the posterior extremities of the sylvian fossa and the parallel or first temporal fissure, and as much as possible of the lateral aspect of the occipital lobe (Figure 13). The aphasic symptoms of a lesion in this region will be dominatingly visuo-aphasic. These will be in the first place word-blindness usually both cortical and subcortical, letter-blindness and number-blindness, alexia, visual agraphia, lateral homonymous quadrant anopsia or hemianopsia.

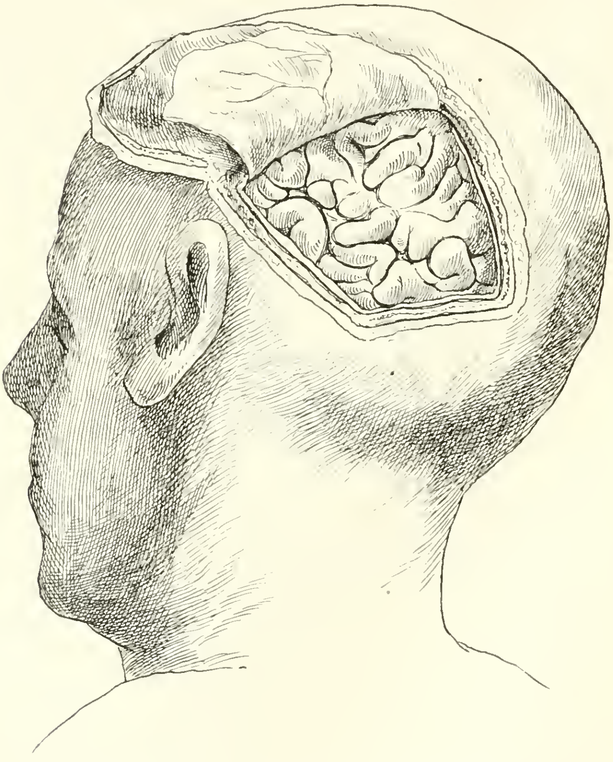

Fig. 14.—Osteoplastic flap to uncover the angulo-occipital region.

In making this statement we accept the view that the anterior portion of this angulo-occipital area is pre-eminently the site of the cortical functions of word-seeing, letter-seeing and number-seeing. The hemianopsia of course results when the lesion invades deeply enough to involve the optic radiation of Gratiolet. Word-dumbness is frequently present, though usually partial. Visual apraxia or mind-blindness is observed in rare instances, but usually only when a unilateral (left-sided) lesion is very large and may involve the commissural fibers of the posterior extremity of the callosum. Mind-blindness like mind-deafness most frequently results from bilateral lesions, and scarcely ever shows itself in complete form except when both sides of the brain are implicated. Inability to copy of course goes with the alexia except in the rare instances of subcortical tumors in which the cortex of the angular region is not destroyed. In such cases the patient may occasionally be able to write, but his eyes once removed from the page cannot, when returned to it, recognize what he has written.

Among the interesting symptoms of lesion extending into the occipital lobe from the angular region are those of color disturbance, especially color amnesia or color dissociation. Wildbrand, Lewandowsky[7] and Spiller[8] have described interesting examples of these unusual occipital symptoms. The patient is unable to name colors which he sees or to pick out colors which are named to him, and yet in some cases at least he may be able to match colors, as with skeins of wool or silk, recognizing their likeness but not being able to designate them by name. Some regard this symptom as a dissociation of color and form sense (Lewandowsky, Spiller). Spiller, for instance, speaks of a case in which the patient was unable to see any incongruity in the picture of a dog colored blue and of a donkey colored green, remarking that he had seen dogs and donkeys which had this appearance. These color disturbances occur when the lesion invades toward the tentorial aspect of the occipital lobe.

Paraphasia may be present in a large angulo-occipital lesion, but is usually partial, and certain forms of auditory aphasia, also partial, like word-deafness, may be present. Anomia or word-dumbness is a comparatively frequent symptom and may be present for reasons already explained when discussing the consequences of lesions of the midtemporal region.

ANGULO-OCCIPITAL OSTEOPLASTIC OPENING

The surgeon in operating on the angulo-occipital region must keep in mind the peculiar shape of the skull as it approaches the occipital pole, the position of the great sinuses and the best places for breakage of the flap. The most approved opening to uncover the angular gyre and lateral occipital lobe as low as possible is, we believe, that shown in Figure 14. As here shown,

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[7] Lewandowsky: Monatschr. f. Psychiat. u. Neurol., 1908, xxiii; cited by Spiller.

[8] Spiller, W. G.: The Journal A. M. A., Dec. 18, 1909, p. 2078

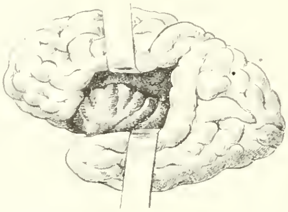

Fig. 15.—External relation of the lenticular zone, the operculum being retracted to show the insula.

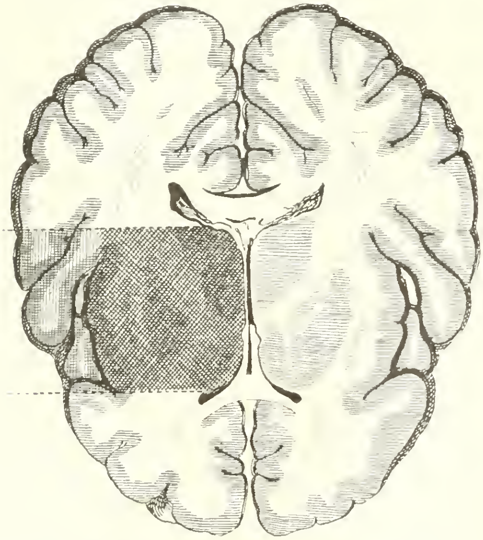

Fig. 16.—Horizontal section of the brain showing deep-seated structures of the lenticular zone on the left corresponding to the shaded area on the right.

the breakage is made to take place at the anterior limit of the opening. Objection may be made that the opening is larger here than at the posterior boundary and about the same size or nearly the same size as at the superior or inferior line. This method of breakage, however, gives the best opening if the surgeon finds that it may be necessary to extend it somewhat inferiorly or posteriorly. One of the doubts sometimes present when an angulo-occipital tumor is diagnosticated is as to how far it is lateral and how far inferior in location, and some opportunity of demonstrating this by exploration is left when the inferior and posterior borders of the opening can be slightly enlarged. Blood-supply is not insufficient wherever the line of breakage may be chosen. It is probably somewhat better when it is placed below. A shorter line of breakage is obtained when this is chosen posteriorly, but it is not difficult for a surgeon experienced with work of this kind to break the skull for any length, especially if he assists in doing this by weakening the flap by means of the Gigli saw. If the short line of breakage is made posteriorly behind the lambdoidal suture the flap may tend to break at this suture and this may cause slight delay and necessitate a second breaking farther back. The opening here described and shown has the angular convolution pretty nearly in its center.

INOPERABLE TUMORS IN APHASIC CASES

Certain aphaso-agraphic cases, especially of tumor, are clearly inoperable. In these the lesion is deep-seated, either in the angulo-occipital region, Wernicke's zone, or the lenticular zone (Figs. 15 and 16). The question of diagnosis is simply one of separating a deep and destructive subcortical from a compressing and penetrating membranocortical lesion. As many cases of aphasia are due to deep-seated lesions of the lenticular zone, it is well to bear in mind the pathognomonic phenomena of such lesions. These are, briefly stated, the completeness of the accompanying hemiplegia with such phenomena as indicate clear involvement of the capsules, internal and external, the claustrum and basal ganglia.

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ETAT VERMOULU—A FORM OF SENILE CORTICAL DEGENERATION.*

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This form of degeneration of the brain cortex to which Marie ascribed the name Etat Vermoulu, or worm-eaten state, has been reported by several of his pupils (Dougherty, Léri, Ficai, Rossbach), but up to the present time has not been described in any of the English or American journals.

For my material I am indebted to Dr. William G. Spiller and Professor Pierre Marie, in whose laboratory I started the work on this subject. I wish also to thank Dr. Allen J. Smith for his kindness in photographing the specimens.

Etat Vermoulu is found in senile brains, particularly in those having a more than usual degree of arteriosclerosis. According to Léri it was present in two per cent. of all the brains examined at the Bicêtre.

It occurs as somewhat irregularly shaped and sized ulcerations on the surface of the brain with the formation in the more advanced cases of small cysts or cavities in the deeper subcortical tissue.

The clinical picture as a rule is not other than that of a senile dementia of greater or lesser inten-

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*Presented at the meeting of the Philadelphia Pathological Society, April 25, 1912.

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sity. Perhaps this is in part due to the frequent occurrence of the disease in the frontal lobes, though the place of predilection seems to be the temporal region. The involvement of other cortical areas, according to the location, may give different symptoms. Often there are some motor disturbances from the presence of additional subcortical lesions, such as small areas of softening (lacunæ) in the internal capsules.

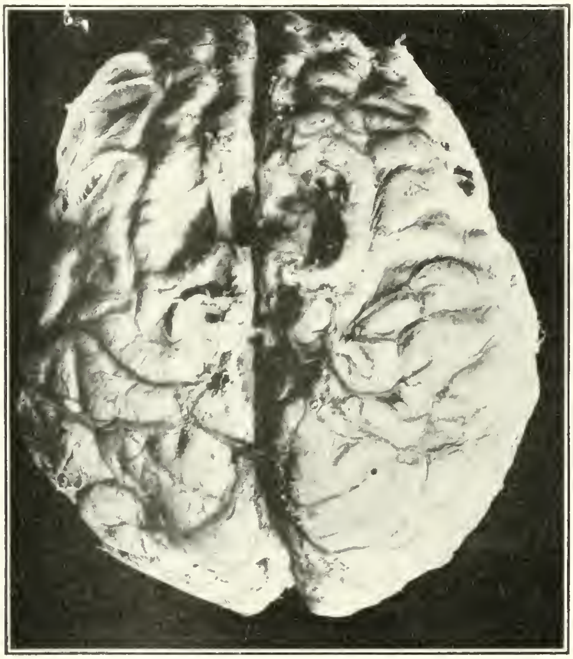

FIG. 1.—View of top of brain showing ulcerated appearance of the frontal and paracentral areas. (Case of Dr. Spiller.)

The gross appearance of such a specimen is that of numerous small ulcerated areas on the surface of the brain. In a fresh specimen they appear yellow, gray, or brownish in color, the walls usually becoming stained by the yellowish fluid or semi-gelatinous substance contained in the cavities.

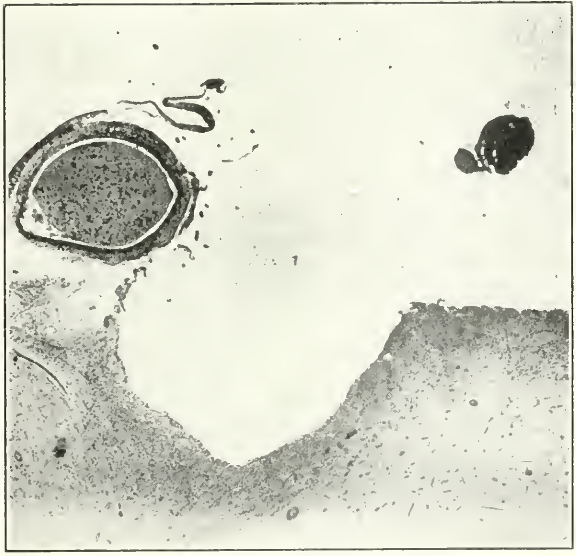

FIG. 2.—Section of the frontal cortex, stained by Van Gieson's method, showing the cone shaped ulceration of the cortex and the glial thickening of the cavity wall. (Case of Prof. Marie.)

These ulcerations are irregular in shape and outline. They have sloping walls and extend only to the border of the white and gray substances. Several adjacent convolutions may be involved or there may be several unaffected convolutions intervening between those more seriously affected. The seemingly unaffected convolutions, however, by microscopical examination show intense changes, probably the beginning of what would later have the characteristic lesion of the etat vermoulu.

The pia, due to its atrophied condition over these areas, usually tears very easily, falling inward and leaving irregularly sized funnel-shaped cavities with the bases outward.

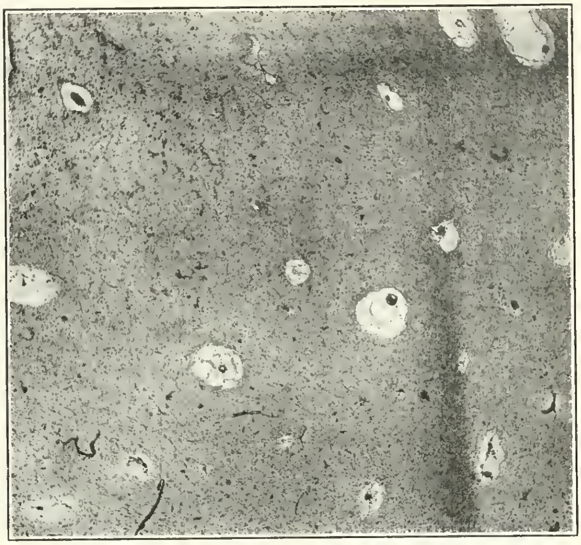

FIG. 3.—Specimen of temporal cortex, stained by Van Gieson's method, showing the intense degeneration around the walls of the blood vessels, forming the large perivascular spaces. (Case of Prof. Marie.)

Microscopically, the pia in close proximity to the ulcerated areas is infiltrated and thickened, the blood vessels are sclerosed, hyaline, and many of them almost completely occluded. Calcareous degeneration, so frequent, according to Ficai, was only present in one or two arteries. Directly over the ulcer the pia is usually thinned, atrophied, and fallen in, forming irregular folds near the edges.

The cortical substance shows a proliferation of the glia, this often taking the place of the tangential fibers as well as of the cellular elements. The glial overgrowth is mainly confined to the fibrillary substance as there appears to be no excessive increase in the glial nuclei. Such overgrowth is probably secondary to the earlier process of softening from the imperfect nutrition following the gradual obliteration of the lumens of the cortical vessels.

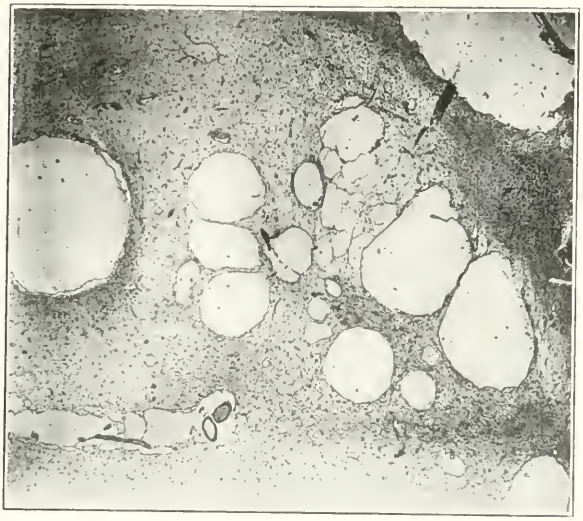

Fig. 4.—Section of temporal cortex stained with hemotoxylin-eosin, demonstrating the great degree of disintegration and vacuolization of the cortex and the absence of any ganglion cells. (Case of Prof. Marie.)

In a number of the sections there appeared to be an unusually yellow tinge in the walls of the ulcerated areas, suggesting the presence of blood pigment. This was probably caused by the absorption of blood coloring matter before its chemical change into hemosiderin. No reaction was obtainable to the tests for hemosiderin, though several vessel walls stained a greenish blue by the ferrocyanide method showing the presence of albuminate of iron in the vessel coats. The possibility of blood coloring matter being carried in solution and not giving the iron reaction before a certain definite chemical change has taken place is shown in a recent article by Claude and Loyez.

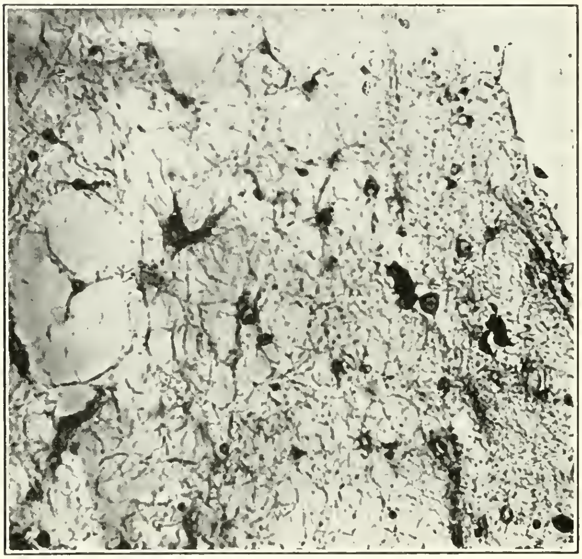

Fig. 5.—Area from Fig. 4 with greater magnification illustrating the absence of nervous tissue and the prominence of the neuroglial reticulum.

Amyloid bodies are very numerous, much more so than in ordinary conditions of senility, and in some of the sections they form almost a solid layer in the region of the tangential fibers. Other bodies having a hyaline appearance are scattered throughout the sections, staining a deep pinkish red by both the hematoxylin-eosin and Van Gieson methods. These are blood vessels occluded by hyaline material so as to form solid hyaline masses, in which there are in many cases no distinct vessel walls visible. Where the longitudinal axes of the vessels have been cut there are long, irregularly shaped hyaline cords.

Throughout the gray cortex and to a much lesser degree in the subcortex, the vessels are surrounded by broken-down, rarefied tissue, leaving in its place only a slight perivascular reticulum of glial fibers, containing a few nuclei, some cellular débris, and fluid. These perivascular cavities may be seen also in the substance of the cord in one of the specimens examined. Such small areas are doubtless points of origin for the cysts occurring in the advanced stage.

The areas or plates of miliary sclerosis described by Redlich and referred to by Léri and others as being frequent in this condition could not be found in any of the specimens examined.

With the extension and confluence of these cavities and the overgrowth of the glial and even, to some extent, of the connective tissue, the cellular portion undergoes destruction in varying degrees. Even in those cases where the cystic formation is absent and only a light grade of superficial ulceration is present, the brain cells show considerable disintegration and destruction, due no doubt to the faulty nutrition so common in the sclerotic cortical changes of senility. In some instances the cortical destruction and overgrowth of glia is so great that there remains practically nothing but a loose meshed network of neuroglia.

The disease most frequently affects the temporal lobes, often the frontal and parietal, and Léri reported even involvement of the cerebellum.

In its early stage this condition probably could not be distinguished from cases of senile arteriosclerosis, of which I believe it to be only a more severe and advanced form. These more severe changes are probably (Léri) due to the imperfect nutrition from the gradually occluded arteries, causing a slow breaking down and disintegration of the tissue around the vessels, with its absorption and an overgrowth of glia, to a certain extent compensatory in nature. In the ordinary senile sclerotic changes described by Alzheimer, however, unlike the etat vermoulu, the nerve cells are disturbed and often destroyed, but there is never a true destruction of tissue with disintegration and absorption and cavity formation.

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Intradural Root Anastomosis for the Relief of Paralysis of the Bladder

And the Application of the Same Method in Other Paralytic Affections

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AND
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INTRADURAL ROOT ANASTOMOSIS FOR THE RELIEF OF PARALYSIS OF THE BLADDER

AND THE APPLICATION OF THE SAME METHOD IN OTHER PARALYTIC AFFECTIONS *

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AND
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Any method of surgical treatment which promises relief or cure of a distressing condition hitherto irremediable by any known treatment, medical or surgical, is worthy of close attention and of adoption after its value has been demonstrated. Incontinence of urine and of feces, especially the former, in cases of traumatism causing complete or partial paraplegia is always a distressing condition, and not infrequently one of danger through the probabilities of cystitis and general infection.

We believe that the following case is the first in which the operation of intradural anastomosis of spinal nerve roots (the first lumbar to the third and fourth sacral) has been successfully accomplished, and in only one instance has it, so far as we know, hitherto been tried. The case, as the history will show, was one of severe traumatism at first causing complete paraplegia, including incontinence of urine and feces. In a few months the paraplegia and accompanying anesthesia were largely recovered from, but the patient was left with an absolute incontinence. Eight months after the operation the patient stated that he could dispense with the urinal, which he had hitherto worn constantly, for a period of twelve hours and that with the assistance of pressure

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* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

over the suprapubic region, partial evacuation of the bladder was possible.

Of course several months are required for physiologic union to take place fully in a case of either peripheral or root anastomosis, but the result in this case so far is most satisfactory. The operation has also shown that no reason exists why nerve-root crossing should not become a valuable addition to our neural surgical procedures, not only for the relief of paralysis of the bladder and bowels of organic origin, but also for obstinate and distressing enuresis of unknown cause. The operation also has a field in some forms of residual paralysis after poliomyelitis and probably in some monoplegias of central or peripheral origin.

REPORT OF CASE

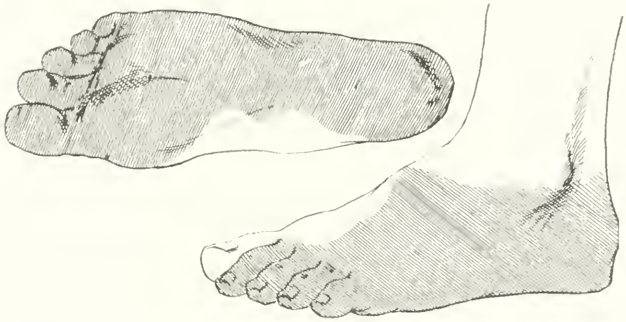
J. S. G., aged 27, married, was a business man who had always had unusually good health until he met with an accident, July 6, 1911. A large gas tank exploded in a shop in which he was at the time. He was thrown some distance with much violence and was rendered unconscious, remaining so for five or six hours. He was taken about thirty or forty miles to a hospital.

He had lost power in both lower extremities. Immediately after the accident his feet were very sensitive to pressure, so much so that it was impossible for him to have his stockings put on. His bladder and bowels were paralyzed. He had to be catheterized for four weeks. He gradually improved, especially as regards power in his lower limbs, but certain very positive conditions remained when he was first examined by us. The loss of power in his lower extremities showed itself then simply in an inability to lift himself and stand on his toes and fore part of his feet, the impairment being therefore chiefly in the calf movements.

His bladder continued paralyzed and in consequence of this he had absolute incontinence, and he had also developed some cystitis. His bowels were torpid, although not so bad in this respect as at first. When they were opened by the use of a cathartic he could not fully control the evacuation.

About a week after the accident he suffered much pain in his back. This disappeared; and he had not had any shooting pains or girdling pains along the spinal column. He had no reflex spasms or jerkings in the lower extremities.

Several examinations gave the following results: Station and gait were good. In attempting to stand on the toes and fore part of his foot he had evident difficulty, although able to perform these movements. For a long time, as above indicated, he was not able to stand in this way at all. Examination of separate movements of the foot showed that he could abduct, adduct, dorsal flex, plantar flex and perform movements of all possible description on the ankle. The power to elevate the heel was slightly impaired. Extension and plantar flexion of the great toe and other toes were preserved. All movements of the leg on the thigh and the thigh on the abdomen were preserved and well performed. Knee-jerk on the right was present and normal; on the left it was much impaired and sometimes absent. Both ankle-jerks were absent. His great toes remained in a slightly extended position but no Babinski response was obtained, and indeed no reflex whatever was obtained on both dull and sharp stimulation of the sole. Examinations for touch, pain and extreme heat and cold showed that these forms of sensation were lost over the sole of the left foot except on an area of its inner aspect, the same being true of the dorsum of the foot, except along its inner border (Fig. 1). The areas of impairment reached about as high as the internal malleolus. On the right foot similar areas were impaired, but impairment or loss was more marked over the toes (Fig. 2). Callus on the dorsum of the right foot at the metacarpophalangeal articulation had been present, according to the patient, since he began wearing shoes after the accident. Sensation was lost over the right buttock and thigh, in an approximately saddle-shaped area, which gradually decreased in width to its termination an inch or two above the popliteal space. On the left a somewhat similar area was present but was narrower and shorter, reaching only to a point just below the gluteal fold (Fig. 3). The scrotum was also anesthetic. An impairment of sensation was present in the coccygeal and anal region.



Figs. 1 and 2.—Areas of impaired sensation on sole and dorsum of left foot.

A study of the areas of anesthesia indicated that the nerve roots or segments of the cord involved were, for the feet, the fifth lumbar and first sacral. and for the buttock, scrotum and anal region, the second and third sacral, probably the fourth and possibly the fifth.

The patient's greatest distress was by far that which resulted from the paralysis of his bladder, with distention and overflow. He constantly had to wear a urinal. He thought that the power to expel the urine had not improved but the inability had probably increased to some extent, while in other respects he was conscious of much improvement. His sexual powers were practically abolished. His feelings of numbness in the feet, and his weakness in trying to stand on his toes he was sure were decidedly less, continuous improvement having taken place. He expressed himself as being quite able to stand all his other afflictions if he could only get rid in whole or in part of the distressing bladder trouble.

The patient was admitted to the hospital of the University of Pennsylvania for further investigation. Roentgen-ray examinations were made by Dr. H. K. Pancoast, who reported a fracture of the right half of the first lumbar vertebra.

After much consideration of the subject with the approval of the patient's brother, who was a physician, and with the urgent approval of the patient himself, it was determined to try to anastomose the twelfth thoracic or first lumbar root to the third and fourth sacral roots. It was recognized that nerve anastomosis offered the only hope for the patient's relief and as this was anatomically impossible outside of the vertebral canal, it was thought that it might be accomplished intradurally.

The operation was performed by Dr. Frazier, Feb. 29, 1912. The following were the notes made by him at the time of the operation and the subsequent hospital records:

Laminectomy—Intradural Anastomosis of the First Lumbar and Third and Fourth Sacral Roots.—Through a curved incision a cutaneous flap was reflected and the spinous processes and laminae of the twelfth thoracic and first and second lumbar vertebrae were removed. Evidently there had been a fracture of the second lumbar vertebra, as several fragments were found quite loose and readily detached. The dural sac seemed unusually distended, and when it was opened a considerable quantity of cerebrospinal fluid escaped (from 1 to 2 ounces), even though the patient was in the reversed Trendelenburg position with the level of the head considerably below that of the pelvis. Inspection of the cord revealed no evidence of injury. There were no adhesions, no areas of softening, in fact no abnormality that could be detected with the naked eye. After consultation with Dr. Mills, I decided to carry out the plan which we had considered carefully before the operation and had submitted to the patient's brother. It was intended to effect an anastomosis between the twelfth thoracic and third and fourth sacral nerves intradurally.

With the assistance of Dr. Edward M. Williams, using a unipolar electrode, the other, a large pad electrode, being closely applied to the shoulder of the same side, the twelfth thoracic and the first lumbar were stimulated within the dural sac, and the one distinguished from the other by the fact that the contractions in the case of the twelfth thoracic were confined altogether to the trunk and abdominal muscles, while in the case of the first lumbar they were associated with contractions of the extensor group of the thigh. Attention was then turned to the conus for the purpose of identifying the third sacral root. What was taken to be the second sacral was first picked up on a blunt hook, and its faradic stimulation was followed by contractions only of the big toe on the affected side. On the ground that we were dealing with the second sacral, the next two nerves adjacent to it and to the inner side were selected for the anastomosis. As it was found by measurements that the twelfth thoracic, even if divided extradurally from its point of exit from the canal, would not reach the conus, it was decided to use the first lumbar. The latter was divided extradurally at its exit from the spinal canal and brought within the dural sac through a transverse incision in the dura. An end-to-end anastomosis was effected by a single No. 1 Chinese silk suture.

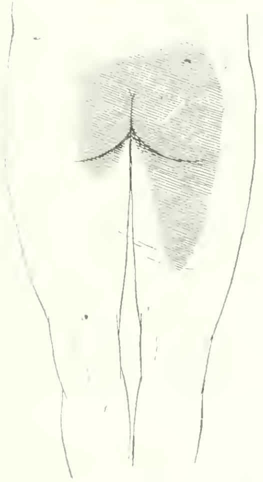


Fig. 3.—Areas of impaired sensation on back of the thighs.

After the nerves had been identified by faradization, pads saturated with a 4 per cent. stovain solution were kept applied to the cord in order to block, if possible, any harmful impulses.

Toward the latter part of the operation the patient's respiration became very shallow and feeble, but on changing his position to his side and resorting to moderate stimulation, normal respiratory function was promptly restored. The dura was closed with a continuous silk suture, and the musculo-aponeurotic layer with buried catgut sutures. The usual cigaret drain, together with a small wick of iodoform gauze, was introduced through a stab wound down to a point of bleeding from the vertebral veins.

The operation was necessarily long and tedious, but at no time were any special difficulties encountered, except in one instance when a vein was divided at the time the first lumbar root was cut. This was followed by quite free bleeding, readily controlled, however, by a tampon.

The identification of the various roots was simpler than had been anticipated. The anastomosis was effected on the left side only, where the sensory evidence indicated less injury to the cord or nerve roots.

On March 4, 1912, the patient complained of deep-seated pain in his thigh, making him gasp. His ankle felt stiff and sore. He thought also he could feel urine accumulating in the bladder.

On March 11, 1912, Dr. Mills examined the patient without moving him from a recumbent position. Flexion, extension and other movements of the thighs were good and all the movements of the feet and toes were present, these having been tested in detail; the only weakness shown was in dorsal flexion and abduction of the right foot, this being only a partial loss. The knee-jerks were absent and there was no Babinski response on either side. Slight plantar flexion of the toes occurred on plantar stimulation.

Tactile sensation, tested by light touch of the finger, was everywhere preserved on the feet and legs below the knees. Pain sense tested by pricking was defective in definite similar areas on both sides. It was lost on the soles of the feet except the inner side about the hollow of the foot half way between the roots of the toes and the heel. On the dorsum it was lost entirely on the inside over all the toes. In a linear area from the metatarsophalangeal articulation three or four inches toward the instep, about the instep and toward the internal malleolus, this area of retained sensation was enlarged. On the outside of the foot a tongue of analgesia extended an inch or two above the ankle. As we were unwilling to move the patient at this time he was not examined over the buttocks, thighs, etc.

On March 27, 1912, the patient was discharged. He felt distention of the bladder on the introduction of 10 ounces of boric solution but he did not feel less amounts of boric solution or of urine.

As stated at the beginning of the article, eight months after the operation the patient could dispense with the urinal.

which he had hitherto worn constantly, for a period of twelve hours, and with the assistance of pressure over the suprapubic region, partial evacuation of the bladder was possible.[1]

NERVE SUPPLY OF THE BLADDER

According to Frankl-Hochwart,[2] whose monograph on the "Nervous Diseases of the Bladder" is among the most reliable and most recent (1906), the bladder has two or three sources of supply. In the first place it has a supply through the sympathetic nervous system. Efferent nerves pass by way of the rami communicantes from the upper lumbar segments of the cord according to Langley and Anderson (at least in the cat). These take their course through the lumbar sympathetic to the inferior mesenteric ganglia. From these inferior mesenteric ganglia three gray hypogastric nerves penetrate the muscles of the bladder. Another sympathetic supply is from the upper sacral segments and roots. These nervi erigentes go to the hypogastric plexus. From this plexus, according to Griffith, the fibers pass in three layers, one to the fundus, another to the anterior part of the bladder and around the neck, and the third to the vesical part of the urethra.

In the human being, according to Frankl-Hochwart and Russian and other authorities with whom he agrees, the somatic nerves to the bladder are from the third and fourth sacral segments and roots.

Kilvington[3] has published the only article on nerve crossing within the spinal canal with which we have become acquainted. In this article he first briefly discusses the fact that regeneration does not take place within the spinal cord, referring to the well-known fact that it seems to be necessary for the primitive sheath to be present in order that such regenerative processes may proceed. He then affirms that such regeneration may occur when nerves are united either intradurally or extradurally within the vertebral canal.

Experiments were made on several dogs in order to test the possibility of bringing about restoration of the functions of the bladder and rectum through nerve crossing and subsequent regeneration. Attention is called to the facts shown by various physiologists, as Langley and Anderson and Sherrington (cited by Schäfer[4]) that in the dog the nerve-supply to the bladder and rectum is chiefly from the second and third sacrals and sometimes from the first sacral and even the seventh lumbar. According to Kilvington the second and third sacral nerves always give bladder contraction sufficient to expel the contents, although the first sacral in a few cases gave bladder contraction, but not enough to cause micturition.

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[1] The patient has reported once since the presentation of the paper—in June—that he is now able to retain urine for two and a half hours at a time and that the expulsive power of the bladder seems a little greater than at the last report.

[2] Frankl-Hochwart, V., and Zuckerkandl: Die nervosen Erkrankungen der Harnblase, Wien, 1906.

[3] Kilvington, Basil: An Investigation on the Regeneration of Nerves, with Regard to Surgical Treatment of Certain Paralyses, Brit. Med. Jour., 1907, i, 988.

Kilvington's instructive experiments on three large dogs and their results are recorded by him in detail. In his summary of these experiments he concludes that it is shown that the last lumbar, when joined to the second and third sacral roots, on electrical stimulation can produce all the effects which the latter give on the untouched side.

After Kilvington had determined by investigation on the cadaver the feasibility of intraspinal root anastomosis in man, Mr. Bird at his suggestion attempted the operation in one case; this operation proved unsuccessful because of conditions found after the laminectomy.

In his experiments on dogs Kilvington in two instances anastomosed both motor and sensory roots (a mixed nerve in effect) to motor and sensory roots below. In another experiment he crossed the anterior or motor root above with both sensory and motor roots below. In the case recorded in this paper we anastomosed a motor root above with a motor and sensory root below.

Certain disadvantages might arise from mixed anastomoses. It is doubtful whether in the surgical procedure the motor root above could be properly apposed to the motor portion of the mixed nerve below. Again, if an anterior root, as in one of Kilvington's experiments, were crossed with both sensory and motor roots below, the only physiologic result, if the procedure were successful, would be the transfer of motor impulses. A motor root of one distribution might physiologically as well as anatomically unite with a motor root belonging to another segment of the cord, but it is doubtful whether a motor root would physiologically combine with a sensory root even if they were brought into physical union.

A sensory root may be combined anatomically and physiologically with a sensory root of its own sort, but

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[4] Schäfer: Physiology, ii, 342.

VENTRAL ROOT ANASTOMOSES

Cervical	Above			Below				Opposite		
..	Cervical	Dorsal	Lumbar	Cervical	Dorsal	Lumbar	Sacral	Dorsal	Lumbar	Sacral
5	..	....	.....	6*	..	.....	.....	....	....	.....
6	5	....	.....	7‡	..	.....	.....	....	....	.....
7	6	....	.....	8§	..	.....	.....	....	....	.....
8	7	....	.....	..	1†	.....	.....	....	....	.....
Dorsal										
1	7*, 8	....	.....	..	2	.....	.....	1*	....	.....
2	8†	1	.....	..	3	.....	.....	1‡, 2, 3*	....	.....
3	..	1, 2	.....	..	4	.....	.....	1, 2, 3, 4	....	.....
4	..	1, 2, 3	.....	..	5	.....	.....	1*, 2, 3, 4, 5	....	.....
5	..	2, 3, 4	.....	..	6	.....	.....	2, 3, 4, 5, 6*	....	.....
6	..	3, 4, 5	.....	..	7	.....	.....	3, 4, 5, 6, 7*	....	.....
7	..	3, 4, 5, 6	.....	..	..	.....	.....	4, 5, 6, 7	....	.....
8	..	5*, 6, 7	.....	..	..	.....	.....	6, 7, 8	....	.....
9	..	6*, 7, 8	.....	..	..	.....	.....	7, 8, 9	....	.....
10	..	8, 9	.....	..	..	.....	.....	8, 9, 10	....	.....
11	..	9*, 9, 10	.....	..	12	.....	.....	8, 9, 10, 11	....	.....
12	..	8‡, 9, 10, 11	.....	..	..	1	.....	9, 10, 11, 12	1	.....
Lumbar										
1	..	8, 9, 10, 11, 12	.....	..	..	2, 3, 4	.....	10, 11, 12	1, 2, 3, 4,* 5	1, 2
2	..	11, 12	1	..	..	3, 4, 5	1, 2, 3	11, 12	1, 2, 3, 4, 5	1, 2
3	..	11, 12	1, 2	..	..	4, 5	1, 2, 3	11, 12	1, 2, 3, 4, 5	1, 2, 3
4	..	10, 11, 12	1, 2, 3	..	..	5	1, 2, 3	10, 11, 12	1, 2, 3, 4, 5	1, 2, 3
5**	..	9*, 10, 11, 12	1, 2, 3, 4	..	..	.....	1, 2, 3	10, 11, 12	1, 2, 3, 4, 5	1, 2, 3

* Barely.
† Easily.
‡ Just.

§ Readily.
** 11 cm. in length.

it may be questionable whether a somatic sensory root will serve to take the place of a destroyed visceral sensory nerve.

Our idea in this operation was to perform an experiment in anastomosis for the relief of the paralysis of the walls or sphincter of the bladder, or of both, similar to that which has been performed extravertebrally in cases of poliomyelitis, traumatic severance of peripheral nerves and other paralyses. The bladder is a muscular organ with a motor nerve-supply, which in large part at least is similar to that of muscles everywhere in the body, although it does in part, as has been shown, receive its innervation through the sympathetic nervous system. By uniting a motor root which is a part of a healthy reflex arc with the motor root of a destroyed sensorimotor region of the cord, both the sensory and the motor portions of the intact arc are utilized. In addition the muscles supplied by the first lumbar nerves in the human subject probably have some accessory value in bladder function.

PRACTICAL SUGGESTIONS

We have discussed the physiologic problems involved in the reestablishment of innervation to the muscles of the bladder, and the operation as performed has been described. Such features as apply only to matters of technic remain to be considered. There is little doubt as to the advisability of a bilateral laminectomy when the identification of roots requires ample exposure and when, as in future cases, we would expect to effect anastomoses between the roots on both sides. It is often difficult to determine the exact position of an individual vertebra, so that in the future we would make sure of this before the operation by having a radiograph taken with a lead strip over what was believed to be the first lumbar vertebra. This would enable the operator at once to be able without the use of the electrode to identify the first lumbar root which leaves the vertebral canal in the foramen between the bodies of the first and second lumbar vertebrae. Therefore the identification of the third or the fourth sacral root would alone remain, and this could be done with any degree of scientific accuracy only by the use of the electrode, as the massed roots composing the conus could not otherwise be distinguished one from the other. It was suggested by Dr. E. M. Williams at the time of the operation that with the bladder filled with boric solution and a small manometer connected with the catheter, stimulation of the third and fourth sacral roots would be followed by an expulsive effort on the part of the extrusor muscles of the bladder which would readily be recorded on the manometer. This is an excellent practical suggestion and will be taken advantage of at the next operation. No difficulty should therefore be experienced in the future, in promptly recognizing the roots to be used in the anastomosis, and needless to say the anastomosis should be effected on both sides and not on one only, as in the case reported.

The principles involved in suturing the divided roots in no wise differ from those which have been laid down for anastomosis of peripheral nerves; it should always be borne in mind that extreme gentleness, the avoidance of trauma, the employment of proper suture material, absolute hemostasis, and the absence of infection are essential to success.

It would be out of place here to discuss in detail the technic of the preliminary laminectomy. It will be sufficient to emphasize two points which we regard as of importance in minimizing the risks of operation. In the first place, we have found the method of administering the anesthetic by intratracheal insufflation to possess great advantages over the drop method not only for safety but also for convenience. We place the patient in these operations in the prone position with the table elevated to an angle of 45 degrees from the waist to the foot, and with the trunk and head in the reversed Trendelenburg position. This position is convenient for the operator and at the same time through the influence of gravity prevents the continuous outflow of cerebrospinal fluid. It is a well-known fact that in prolonged operations when the patient is in the prone position (face down), the respiratory apparatus seems to wear out more readily, and under ether anesthesia proper oxygenation of the blood is interfered with. The use of intratracheal insufflation entirely overcomes this difficulty and an even state of anesthesia is preserved, with adequate oxygenation whether or not the respiratory function becomes fatigued.

The second point to which we refer has to do with the harmful influence of the manipulation of the roots incidental to their identification, isolation, division and suture. There is no doubt that the mere manipulation of the roots originates afferent impulses which of themselves have a harmful influence on the vasomotor system. To break this influence and thereby prevent shock-producing tendencies, we have been in the habit of establishing a block by applying 4 per cent. stovain solution to the cord just above the site of our manipulations. These two points we feel assured are worthy of consideration in rendering an operation, which we believe should be without fatality, free from any immediate depressing influences.

Leaving aside these essentially practical considerations, we may enter for a moment into the purely speculative field. That physical union between the first lumbar and third sacral roots may be effected within the dural sac has been demonstrated on the living subject, and its application to residual paralyses of the bladder following cord lesions discussed. The question at once suggests itself as to whether or not this operation may not be applicable to paralyses of other groups of muscles and we have determined from observations on the cadaver that those anastomoses may be considered possibilities which are indicated in the accompanying tabulation.

From this table it is readily seen that in the roots which go to make up the brachial plexus it would be possible to effect an anastomosis only between the root immediately above and the root immediately below either the fifth, sixth, seventh or eighth cervical. In the case of the first thoracic root, an anastomosis could be effected with the seventh or eighth cervical above and the first thoracic on the opposite side, and in the case of the second thoracic with the eighth cervical and first thoracic above and with the first, second and third thoracic on the opposite side. In cases of injury to the cord involving the seventh or eighth cervical segments with resulting paralyses it would be possible to innervate the roots from these segments with the first thoracic.

The farther down the cord one goes the longer is the distance from the point the root is given off the cord to its point of exit from the canal, and the more the roots overlap one another. When we come to the lumbosacral region we find that the ventral roots lend themselves to innumerable possibilities of anastomosis with roots above or below and even with roots on the opposite side. It would seem quite within the range of possibility in a case of unilateral infantile palsy of the lower extremity to be able to innervate the root of the paralyzed group with a root that might be spared from the same or the opposite side, as has been suggested earlier in the paper.

In the paralyses of the bladder and rectum following trauma, however, we have perhaps the most important indication for this intradural operation, not only because of the patient's otherwise hopeless condition, but because those so afflicted are utterly miserable and their lives are shortened by the infection of the kidneys which sooner or later follows the inevitable cystitis. If this new plan of procedure had no other application than for the relief of the vesical palsy it would still have a wide and important field. That it is based on a reasonable hypothesis has been proved by experimental observations on the lower animals; that it is a justifiable risk no one will gainsay; that it gives promise of attaining its object has been demonstrated by the fact that our patient has already gained some control of the bladder. Owing to the comparatively short time which has elapsed between the time of the operation and the present, this contribution takes on the nature of a preliminary report. We therefore submit it as such for consideration and discussion.

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PARANOID TYPE OF INSANITY WITH JACKSONIAN CONVULSIONS: SYPHILITIC CEREBRAL PACHYMENINGITIS; THE MICROSCOPIC FINDINGS

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PARANOID TYPE OF INSANITY WITH JACKSONIAN CONVULSIONS: SYPHILITIC CEREBRAL PACHYMENINGITIS; THE MICROSCOPIC FINDINGS *

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F. C., a colored man aged 42 years, was arrested Sept. 5, 1910, while sleeping in a new school building, but because of his strange statements he was taken to the psychopathic wards of the Philadelphia General Hospital; from there he was transferred to the Insane Hospital, September 9, where he developed jacksonian convulsions and died about three months later. The cause of his mental state was not determined clinically.

For some time previous to his detention and until he died, certain fixed delusions persisted in the mind of this patient. He stated that the new school building in which he was arrested had been fixed up for him by God's orders and that it was to be called God's Angel's Temple. He drank at times but he had not been drinking for several months previous to his detention at the hospital. About a week after he had stopped drinking, he said he heard God call his name and say: "You are free from sin; you shall live and never die; you shall never go hungry and you shall never go naked." Because of his shouting his friends thought he was crazy from drink and he was arrested; shortly afterward he became quiet and he was released. After this "call" from God he stopped work. He said God talked to him both day and night and told him secrets; but what these were he would not reveal.

His mental examination at this time showed no disorientation; he had hallucinations of hearing, believing that he heard God's voice; his delusions were shown by his belief that the schoolhouse had been built for him by God's orders and that he was God's angel. He stated that he did not have enemies.

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* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

* From the Laboratory of Neuro-Pathology of the University of Pennsylvania and the Philadelphia Hospital for the Insane.

was not possessed of much physical strength and was not wealthy; in fact, he talked sanely about all other matters.

The points of interest in his physical condition were: unequal pupils, the left being slightly the larger; slight tremor of the muscles about the mouth; deviation of the tongue slightly to the left; enlargement of the inguinal glands, and several scars over the shins.

While talking to his physician, Jan. 13, 1911, he said: "I am going to have a fit; I had two of these last night;" he then had a seizure. There was involvement of the left side of the face and left arm without any disturbance of consciousness. The convulsions of the night before were his first seizures. After this he had several isolated convulsions and on April 3, was seized by a series of fits, which involved the whole left side of the body. At one time he had 200 convulsions in sixty hours and these were followed by a left-sided exhaustion paralysis. The seizures were for the most part confined to the left side and were clonic in type, but they were never attended by the loss of consciousness. A warning always preceded them and this the patient described as a peculiar sensation in his head. Plantar stimulation at times gave a Babinski response, which was present more frequently on the left side. The ophthalmoscopic examination showed no abnormality, and there were no extra-ocular palsies, no persistent headache and no vomiting. The patient was at no time melancholy or violent, and his fixed delusions persisted until he died. The seizures usually appeared in series and were followed by a slight elevation of temperature; the last series occurred four days before his death; for a few days previous to this his heart action was irregular, his pulse was over 100 and his temperature was subnormal.

Because of the exalted delusions there was at first some thought of the psychosis being dementia paretica; paranoia also was considered. Later, when convulsions developed, it was suspected that the condition had a syphilitic basis; unfortunately the cerebrospinal fluid was not examined nor was a Wassermann test obtained and so no positive mental diagnosis was made.

AUTOPSY REPORT

The general body findings were: heart, chronic interstitial myocarditis; lungs, passive congestion and subacute bronchitis; liver, fatty degeneration, slight perilobular fibrosis and chronic interstitial hepatitis; spleen, chronic interstitial splenitis; kidneys, chronic interstitial and chronic parenchymatous nephritis. These chronic findings were not more than suggestive of syphilis.

Under the calvarium, the dura in the right superior parietal region was adherent to the bone; when separated, the bone was found, roughened and reddish in color. There was no evidence of an old fracture. At this site and even extending somewhat below the right parietal region, the dura was adherent to the pia-arachnoid, and the superficial part of the cortex was stripped off with the membranes. The area of most extensive pachymeningitis was about 2½ inches in diameter and one-third inch thick near the center, becoming thinner gradually toward the periphery. There was very slight thickening of the dura for considerable distance beyond this, but the rest of the membrane was of normal thickness and texture. The surface of the lesion was somewhat irregular though not distinctly nodular; its consistency varied, being considerably firmer in some parts than in others. The pia-arachnoid was somewhat congested. Beneath the area of most extensive pachymeningitis, the convolutions were somewhat flattened. The base of the brain and the vessels were normal. There was slight excess of cerebrospinal fluid, especially in the lumbar region of the cord. The weight of the brain and part of the membranes which happened to be attached was 1.372 gm. (48.4 oz.); this is but little less than the weight of the normal adult male caucasian brain, and about 200 gm. (6.1 oz.) heavier than the average weight of the brain of the colored adult in dementia paretica.

HISTOLOGIC FINDINGS

The dura was thickened to perhaps three times its normal diameter but it was difficult to determine just where the dura stopped and the extra development of fibrous tissue began. Many irregularly rounded holes were seen and these probably had resulted from the hardening of edematous tissue. The development of fibrous tissue was very extensive and many of the fibers were markedly swollen. Throughout, there was a rather free leukocytic infiltration with here and there intense localization. Embryonic cells were abundant and increased vascularization was observed. Some masses of granular detritus were seen here and there, and these were probably disintegrated red blood-cells.

The cellular infiltration consisted for the most part of three varieties of cells and these appeared to be lymphocytes, mast cells and plasma cells. The lymphocytes were greatly in preponderance. In some areas there were many large, round or oval, highly granular cells; their diameters ranged from 10 to 18 microns. They mostly contained relatively large, round or slightly lobulated nuclei which were usually eccentrically placed. When stained with theonin, these cells took on an intense blue color but under artificial light they assumed a deep purple tint, while the lymphocytes remained blue and the red blood-cells showed a greenish tinge. These cells conformed more nearly to the type of the mast cell than any other with which I am familiar. The third variety of cells corresponded to the description usually given of plasma cells; they were much less numerous, usually larger and tended to be more angular than the mast cells and they contained finely granular protoplasm. In most of the sections, particularly those stained with hematoxylin, many endothelial cells were observed; these, perhaps, were due to active proliferation, but at no time were such cells found within the lumen of the vessels; often they appeared to be engaged in the formation of new blood-vessels.

The pia-arachnoid was much thickened and densely adherent to the dura. Within the meshes of the membrane there was a diffuse cellular infiltration; the vessels were increased in number with some perivascular cellular areas.

There was subpial neuroglial proliferation amounting to a dense felting, and this accounted for the superficial layers of the cortex being removed with the membranes. These neuroglial cells were very numerous and of gigantic size with many hair-like processes radiating from them. Almost all stains revealed the presence of these bodies, but they were most intensely stained by Mallory's neuroglial stain and Weigert's myelin sheath stain. The cells nearest the pia were so closely interwoven as scarcely to be recognized, but as they extended farther into the cortex, their outlines and processes became clearly visible. In contrast with the commonly seen stellate glia cells, their sectional area was four or five times as great; they were mostly oval and had many exceedingly long processes; they frequently contained two or more nuclei; they were proliferating most rapidly and were seen in all stages of development.

The cortex immediately beneath the involved meninges showed some perivascular cell infiltration, but this probably had extended from the meninges. A most destructive process had gone on in the superficial cortical area. Some of the pyramidal cells were barely recognizable; many were shriveled up; others appeared as a pale blue area in which nothing definite could be observed except the nuclei; the angles of the cells were almost lost and the tigroid bodies in many places had disappeared entirely; sometimes the nuclei were not apparent, frequently they were vesicular, often they were displaced and occasionally they bulged beyond the cell outline.

The most superficial medullated fibers showed some varicosities and attenuation but elsewhere the fibers were normal. The vessels at the base of the brain were normal.

There is no specific character to a syphilitic psychosis; it may partake of the type of dementia, mania, melancholia or paranoia. Of course the same is true of an alcoholic psychosis, and since this patient drank to some extent, it is possible that alcohol played a part in causing his mental condition; but this certainly did not account for his convulsions. As to the pachymeningitis which doubtless was the cause of the convulsions: It has been said that syphilis "never attacks exclusively any small area of the brain or cord, it is never strictly local, but always more or less generalized." There was no history of traumatism in this patient nor was there any evidence of an old injury in the bone over the area involved; still, in a syphilitic subject, the possibility of traumatism causing such a condition is pointed out by Mott[1] who has met such cases following "a blow on the head which has set up an inflammation either in the bone or in the dura mater at a definite spot just as a blow on the shin may cause a periosteal node in a person the subject of syphilis."

In this case a clinical diagnosis had not been made and at necropsy the growth was not sufficiently distinctive to reveal its true nature. Microscopic examination of the meninges and brain showed no distinct abnormality except in the areas described; this ruled out dementia paretica; the excessive weight of the brain was likewise against this condition.

The microscopic findings conformed to those found in syphilitic pachymeningitis.

I am indebted to Dr. J. Allen Jackson of the Philadelphia Hospital for the Insane for the opportunity of studying this brain.

2005 Chestnut Street.

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1. Mott: A System of Syphilis, iv, 81, Oxford University Press, London, 1910.

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MUSCULAR HYPERTROPHY WITH WEAKNESS*

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Overtrained athletes and overworked laborers with physiologically hypertrophied muscles may show distinct loss of power. This weakness is temporary, and strength is restored by proper rest. There is, however, a condition of pathological hypertrophy of muscle in which is found pronounced and permanent loss of power. Naturally such cases are at first considered pseudo-hypertrophic dystrophy, until histologic examination of excised muscle shows the absence of degeneration and of fatty or fibrous overgrowth.

Cases of true muscular hypertrophy with diminished power have been described as early as 1875 by Friedrich and Auerbach, The latter[1] found in a soldier 21 years old marked increase in circumference of certain muscles, increased thickness of individual fibers but no proliferation of fat or fibrous elements, and no degeneration. In this man only the right upper extremity was affected. In this limb the patient had first noticed increase in size together with early fatigue in any prolonged effort. The fatigue increased until the usefulness of the member was much reduced. The affected muscles felt harder when contracted than normal muscles. In a single effort considerable power could be exerted, but the muscles soon became tired and the contraction weak. The skin over the affected muscles was of a bluish, mottled color, and felt cooler than that of the other arm.

Examination of excised fibers showed a fiber diameter of 75μ to 175μ, most of them being between 110μ and 145μ. Corresponding measurements in the left limb showed a maximum of 104μ, most lying between 58μ and 75μ. All the muscles of the right upper extremity were involved, but no other muscle in the patient's body.

In the cases reported by Friedrich[2] the same enlargement of muscle mass depending upon truly hypertrophied fibers, without fatty or fibrous change and without degeneration, is described. In these patients the subsequent course was followed and no reason found for considering them merely early stages of pseudohypertrophic dystrophy.

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A series of cases described by M. Bernhardt[3] as "Muscle Rigidity with True Hypertrophy" were, I suppose, really to be classed as cases of Thomsen's disease.

Sakaki[4] reported a case with true hypertrophy of the muscles of both left extremities, with marked weakness, no degeneration, no fibrous or fatty overgrowth, but with choreiform movements. The patient was 14 years of age. His speech showed muffling of articulation, and scoliosis of the vertebral column was present.

Schoenborn[5] has recently published a case of hypertrophy of the tongue and upper arm muscles in which the same early fatigue was manifest. The man was 44 years old. Simultaneously there had been swelling of all the salivary glands. The excised bit of muscle showed increase of nuclei, but no degeneration; and subsequent observation of the patient showed no change in the muscular trouble.

Eichhorst's observations[6] on this condition are set forth under the caption "Hypertrophia Musculorum Vera": The essential conditions present are increased external circumference of the limbs through gross enlargement of the muscles, with enlargement of the individual fibers, without proliferation of connective tissue or fat. He suggests that the cause of the trouble may be embryonic fault in the muscle cells, to which trauma, overuse and infectious diseases may act as accessories. Males are most commonly affected. In explanation of the defective power, this writer proposes faultiness of the nerve end-plates, or insufficiency of blood supply for the increased muscle mass (relative ischemia). His description of the course and symptoms of the disease is as follows: The disorder usually develops gradually. There may be neuralgiform pains, though sensory disturbances are rare. The upper limbs are most often attacked, next the lower limbs, then the shoulder girdle. The condition may be unilateral. The points that most impress the patient are the athletic appearance of his muscles and the paradoxical weakness in continued effort. Berger described a case in which fibrillary twitching occurred. Electrical reactions in most cases were normal, in a few they were decreased. Benedikt found mechanical excitability increased. Vasomotor disturbance with discoloration of the skin was occasionally seen. Eichhorst considers the condition an independent pathological entity.

A patient examined in the University Neurological Dispensary in 1909, and observed from time to time subsequently, is probably a victim of this disease. As there are several interesting points in his history and present condition which raise nice questions of diagnosis, the following description may repay perusal.

William B. is a black negro laborer, grandson of a pure-blooded African immigrant, 47 years old (in 1909), whose father died of phthisis. A brother is a hemiplegic in the Philadelphia General Hospital. No disease of the muscles is known in any branch of the family. The patient is the youngest of a family of ten. He denies syphilis, though he admits having had gonorrhea six years ago. Of his wife's twelve pregnancies, the last aborted and five children died in infancy.

When the patient was 17 years old and in good health, he found on awaking one morning that his whole left side was paralyzed, the skin of that side being hyperesthetic. His memory is vague as to details of this seizure, but he is sure it cleared up entirely, so that after three years no weakness remained. As a youth he was of unusual muscular development, and he cites specific compliments from employers upon his fine build and great strength. Until his fortieth year he worked at the hardest forms of manual labor.

The first dawning of his present trouble came six years ago, when he noticed that he was not as strong as before; gradually he dropped from the van to the rear of his gang in digging and lifting, and step by step has degenerated into a mere house- and lawn-worker. He sought help in the dispensary also because of weakness of the posterior neck muscles which caused marked bowing of his head, and because of general muscular pains. Examination then showed splendid development of the muscles of arms, forearms and back, and his general appearance was suggestive of fitness and power, though the lower limbs were not as massively developed as the upper. All muscles felt firm when contracted. There was lordosis of the lumbar spine, the concavity being emphasized by the overdevelopment of the long back muscles.

No fibrillary twitching was seen; but one of the patient's chief complaints was of painful muscular cramps. While he was being examined one of these cramps was seen: a small mass of muscle 2 to 4 cm. in diameter rose up, became hard and formed a rounded tumor, which remained a moment and subsided. There was no undue contraction of muscles at the beginning of voluntary movement, though soreness of the muscles made the beginning of movements slow and cautious. Faradic and galvanic stimulation showed no marked change, the contractions being possibly over-prompt and over unusually extensive areas. Even light tapping of the muscles caused prompt contraction of fibers; in the deltoid and biceps of each side this amounted to a jerk of the whole muscle. There was no myasthenic reaction.

In addition to these symptoms there is occasionally some stumbling in speech, and he complains of choking during meals. He wets his clothes if delayed in reaching the urinal; the rectal sphincters do normal duty.

Tendon reflexes are unduly prompt in all four limbs, there is no spasticity, and no ataxia. His station is good, the gait good on first starting out, but soon becomes unsteady from weariness. His plantar reflexes are normal. Eye movements and pupillary reflexes are normal. There is no nystagmus and no tremor. His finer finger movements, as in buttoning shirt, are awkward.

To avoid inaccuracy in estimating the patient's strength, I asked Dr. MacKenzie to put him through the usual tests of the university gymnasium. The results follow:

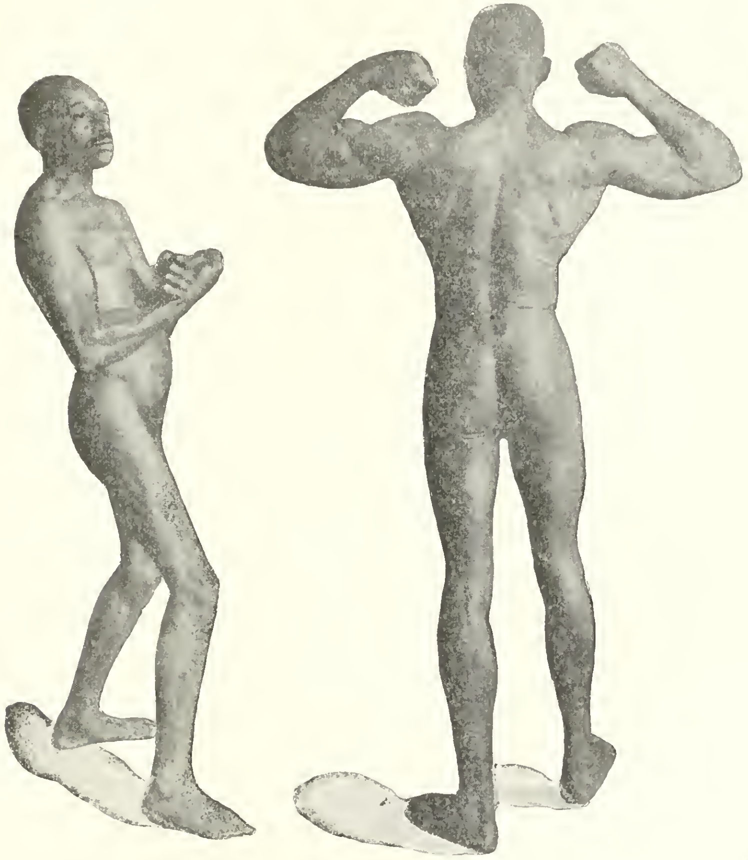
	Wm. B.	Average Student.	An Untrained Middle-aged Man.	Athletes
Lifting by extension of thigh and leg	180 kg.	245 kg.	300 kg.	530 kg.
Lifting by extending trunk on thighs	120	120	170	530
Flexion of upper extremities	100	105	140	185
Extension of upper extremities	120	110	160	210
Grip, right hand	44	44	60	70
Grip, left hand	45	40	60	64
Totals	609	664	890	1,329

The patient, although a laborer and of unusual muscular development, falls below a man leading a sedentary life, below even the averages of 2,000 students amongst whom were not merely the young and sedentary, but even some sickly individuals. He falls far below the athletic type of student, with whom in these tests he ought to compare favorably, the tests being not such as require knack, but those which laborers should best execute. The strength of each limb was practically equal to that of its fellow of the opposite side.

In endurance he is particularly faulty. He can walk several miles, but after the first half-mile his gait becomes unsteady, even staggering, and he feels great weariness. The only sensory symptoms were occasional dull muscular pains, which appeared to trouble him but slightly. Tactile, pain and temperature perception was normal.

Microscopic.—A piece of muscle excised from the left triceps shows distinct enlargement of fibers in cross section. The fibers are nearly round in cross-section, the diameters ranging between 70μ and 180μ; most of them falling between 130μ and 160μ; the average being 130μ.

Fibers of muscle tissue taken from cadavers are somewhat smaller than those removed from living muscle, and they range in diameter from 10μ for small muscles of the eyes, up to 100μ in the large muscles of trunk and limbs. Fibers of muscle removed during life and fixed at once in Müller's fluid, and used as controls in my measurements gave 108μ as maximum diameter, most diameters lying between 60μ and 80μ.



In Wm. B. the sections showed beautifully clear longitudinal- and cross-striation, the fields of Cohnhein being very distinct.

There are two peculiarities shown by the nuclei: some fields had a greater number than normal of endomysial nuclei, without, however, there being any increase of endomysium. The nuclei appear to lie upon the sarcolemma. But where fibers had been somewhat separated from each other, a very thin band of connective tissue could sometimes be discerned, to which the nuclei in question adhered. The long, cylindrical muscle nuclei were not increased in number, but a considerable proportion of fibers had the nuclei deeply placed within the cell, and not immediately beneath the sarcolemma as is usual. Nineteen per cent. of the fibers had from one to four nuclei thus situated.

There was no increase in fibrous tissue, endomysium and perimysium being as in normal fibers. There was no sign of fatty overgrowth, and no degeneration of muscular tissue. No round-cell infiltration appeared.

The diagnosis first made, of pseudo-hypertrophic muscular dystrophy, seems improbable. The man is a black negro, appearing and claiming to be pure-blooded.[7] The disease commenced after he was forty years of age, and there is no family history of muscle disease. These points do not, however, exclude dystrophy. The insurmountable difficulty is that there is no atrophy, macroscopic or microscopic. His tendon reflexes in even the muscles most involved are not subnormal, but rather increased as are galvanic and faradic excitability.

There is not the soft, flabby feeling to this man's muscles which characterizes muscular dystrophy, nor does microscopic examination show any sign of fatty or fibrous overgrowth. Our patient shows, moreover, certain symptoms not to be expected in pseudo-hypertrophy: painful cramps, dribbling of urine, dull pain when the muscles have been rested—dystrophic muscles may give pain after overuse—and abnormal mechanical and electrical excitability.

Examination made in February, 1911, after the above description was written, shows some improvement in patient's general health, muscular power unimproved, but not less than two years ago. He complains less of the painful cramps except in the great toe of one foot, which has extension cramp more painful than before, and more frequent. He holds his head more nearly erect. There is no spontaneous muscle pain, but slight squeezing of the enlarged muscles gives marked pain. Otherwise his condition is unaltered. The affected muscles when contracted feel as firm as those of an athlete. This absence of atrophy during eight years in which the disease has been present makes the diagnosis of pseudo-hypertrophic disease more improbable.

Thomsen's disease could not be present without the tonic contraction at the beginning of voluntary motion, which is its essential symptom. Nor do the muscles present the board-like hardness usually found in that disease. Similarly myasthenia gravis cannot be thought of since there is no myasthenic reaction to faradic stimulation and the gradually developing fatigue is very different from the rapid exhaustion of myasthenia.

The purpose of this report is not, however, to debate the diagnosis of a particular case, but to call attention to a disease of the muscular system the characteristics of which are as follows:

Increased bulk of affected muscles, which feel firm when contracted; diminished power with early fatigue; hypertrophy of individual fibers, some increase in number of nuclei; no proliferation of fibrous or fatty tissue, and no degeneration; tendon reflexes, mechanical irritability and electrical reactions varied from slight diminution to slight increase. No qualitative electrical changes; a few cases show vaso-motor skin changes; and some, dull pain or tenderness of muscles; the course is slow, but little change occurring from year to year; males are more often affected. The upper extremities were involved more than the lower, though any part of the body may suffer.

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DISTURBANCE OF SENSATION IN A CASE OF SYRINGOMYELIA[1]

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It is impossible for one to scrutinize the work on the centripetal tracts of the spinal cord from 1846 to the present without realizing that the diametrically opposite results and conclusions arrived at by the long list of investigators have caused "confusion worse confounded."

The study of the centripetal systems received its greatest impetus in 1846 when Brown-Séquard published his historic thesis (1). The value of his thesis is purely relative and it takes high rank only when considered from the standpoint of its early date. The most interesting observations that he makes at this time are the following:

(P. 25.) "5° J'ai coupé les faisceaux antéro-latéraux: il y a une paralysie complète du mouvement volontaire dans les parties qui reçoivent leurs nerfs de la portion de moelle postérieure à la section. La sensibilité est restée intacte."

"6° J'ai coupé les faisceaux postérieurs: le mouvement volontaire reste à peu près intacte chez les grenouilles et les anguilles; il est diminué chez les autres animaux (pigeons, lapins, chiens). Le sentiment subsiste sans grande diminution dan les parties placées en arrière de la section."

(P. 26.) "Après avoir coupé le faisceau antéro-latéral et le faisceau postérieur d'un côté de la moelle sur des grenouilles, des pigeons et des lapins, j'ai trouvé les parties situées en arrière de la section et du même côté aussi sensibles en apparence que celles du côté sain. Quant au mouvement volontaire, il était évidemment diminué; mais je n'oserais pas déclarer qu'il n'existait absolument plus."

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[1] Read at the thiry-sixth annual meeting of the American Neurological Association, May 2, 3 and 4, 1910.

The lack of value of many deductions at this time is aptly evidenced by the following:

(P. 29.) "Je dois signaler un autre fait important, demontré par mes expériences, c'est la facilité avec laquelle les impressions sensitives se transmettent d'un côté à l'autre de la moelle. Ce fait explique pourquoi, dans les hémiplégies, la perte du sentiment, et surtout la perte compléte, est si peu fréquente."

An adequate concept of the chaos in the knowledge of the ascending and descending tracts can be obtained by a perusal of Brown-Séquard's lectures (2), in which the most grossly flagrant statements as to function and anatomy pass unchallenged by him.

In the Experimental Researches applied to Physiology and Pathology (3), Brown-Séquard describes the paralysis which has ever since borne his name. This paralysis concept has needed much alteration and revision, which it obtained at the instance of Brown-Séquard himself. And to-day we find this concept very vulnerable in several particulars.

There are several difficulties met with in attempting a solution of the question of the pathway of centripetal impulses in the spinal cord. The contradictory results obtained in animal experimentation, particularly on the part of the older investigators, attract attention. A great deal of the evidence gleaned by these older operators must be discounted owing to the grossness of the technique which then obtained. I see no reason why many of the old-time experiments should not be repeated with advantage, modern technique and asepsis offering so much better end results. For instance, some of the early investigators made median longitudinal sections of the spinal cord in animals and reported complete paralysis, motor and sensory, of the limbs below the line of action. I have repeated this work in dogs, making median longitudinal sections of the cord in the thoracic and lumbar regions. The dogs have shown no motor weakness or difficulty in walking after coming out of ether.

We are then confronted by another question which has caused much confusion: in lesions of the spinal cord corresponding in extent to a hemisection, there is much difference in observations as to the alteration of tactile sensation of the homolateral and contralateral sides. The question of pain and temperature conduction in the spinal cord does not give much trouble, it being conceded by most that the fibers of the posterior roots bearing these impulses penetrate the posterior horn of the same side and make synapse with an ill-defined group of cells lying external as well as anterior and posterior to the Clarke vesicular column. The axones from these perykaria cross over to the opposite side of the cord via the anterior white commissure, as stated by von Bechterew (4). The further course of these axones is in the antero-lateral column, in or near Gowers' tract. It is doubtful whether any of these fibers are tautomeric as has been suggested by Barker (5).

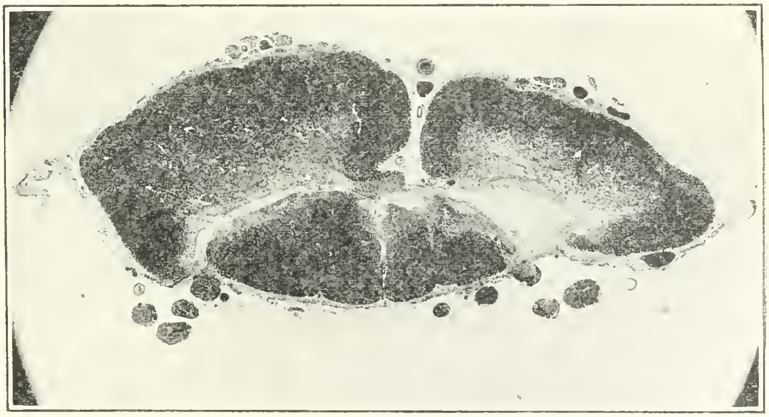

Fig. 1. Eighth Cervical Segment. Weigert Preparation.

Naturally with this arrangement of fibers the hemisection of the cord will cause analgesia and thermo-anesthesia of the contralateral side.

As to the afferent spinal pathway for muscle sense, there is no controversy, it being admitted that the fibers subserving this sense pass up in the posterior column of the homolateral side.

But the most difficult point to explain in the Brown-Séquard type of paralysis is the strange inconstancy of tactile anesthesia. In some cases, chiefly those observed years ago, there seems to have been, if correctly observed, total loss of tactile sensation on the contralateral side. As the technique of differentiation between the several forms of sensation grew more delicate, we find in the reported cases of Brown-Séquard symptom complex the fact noted that the loss of pain and temperature senses on the contralateral side was complete, but that the tactile sensation was only slightly, if at all, disturbed.

I quote herewith from Dejerine (6) to show the trend of observation on this point:

"Les phénomènes croisés consistent en une anesthésie totale du membre inférieur. Une anesthésie aussi complète s'observe moins fréquemment qu'on l'a dit: dans un nombre considérable d'observations, il ne s'agit que d'une diminution des différents modes de la sensibilité, beaucoup plus accentuée pour les sensibilités thermique et douloureuse; il existe alors dan ce cas une véritable dissociation syringomyélique. Dans les hémiparaplégies à évolution lente, par tumeur, gomme ou tubercule de la moelle, l'anesthésie s'établit progressivement et dans l'ordre suivant: anesthésie thermique, douloureuse, tactile; la sensibilité tactile sans être abolie, peut être très altérée; il existe des erreurs de localisation, une diminution de la perception, appréciable seulement au compas de Weber."

It is to my mind a trifle questionable whether we should consider errors in localization or diminution of sensation only appreciable by Webber's compass correctly classed with tactile anesthesia.

Spiller reported a case (7) in which the antero-lateral column on each side of the spinal cord was involved by a well-defined tuberele. On the right side the tubercle was at the level of the twelfth thoracic segment, on the left side it was from a half inch to an inch higher. There was loss of pain and temperature senses but preservation of tactile sense.

There being no appreciable decussation of fibers in the posterior columns, and allowing that the tactile impressions are conducted in the posterior column of the same side: how is it that with a hemisection or a lesion equivalent thereto one should have usually a preservation of tactile sense on the homolateral side?

In elucidation I can not do better than quote the following from Sacki and Schmaus (8):

"Grössere Schwierigkeiten macht uns die Erklärung der Thatsache, dass der Tastsinn oft keine oder nur geringe Störungen zeigt; eine recht plausible Erklärung dafür hat Mann gegeben. Er geht davon aus, dass ein jeder Reiz, der eine spezifische Empfindung auszulösen pflegt, wenn man ihn sieh nur genügend vermindert denkt, schliesslich nur noch eine Tastempfindung hervorruft. Nimmt man nun auch eine direkte Bahn für die Tastempfindung in den Hintersträngen als vorhanden an, so kann doch diese Empfindung auch wenn ihre eigene Bahn unterbrochen ist, auf jenen anderen, für Schmerz, etc., als Minimalempfindung den höheren Centren noch zugeführt werden; eine Tastempfindung kann also immer dann noch zu stande kommen, wenn nur irgend eine centripetale Bahn im Rückenmark noch erhalten ist."

It were well to remember that a sensory nerve fiber, be it for temperature, pain, tactile or other special form of sensation, possesses specificity for that particular form of stimulus in that that specific threshold is lowered and other thresholds relatively raised. Mann's idea (9) is simply that when the specific pathway for tactile sensation is cut, that any other afferent pathway may be used in carrying the tactile impulses.

Therefore, as an example, in left hemisection of the cord, allowing that tactile stimuli pass up the posterior column of the same side, we do not have tactile anesthesia of the homolateral side because the other afferent pathway passing up the anterolateral column of the contralateral side is brought into requisition.

The question which is still left unanswered is: if the pathway for tactile stimuli lie wholly in the posterior column of the homolateral side, how can one account for tactile anesthesia of the contralateral side in hemisection or equivalent lesion?

In answering this question I wish to call attention to the following points:

(*a*) If tactile impressions pass up the posterior column of the homolateral side, exclusively, it is easily seen that of the several forms of sensation the pathways of tactile sense for each side of the body lie closer together than do the pathways for any other form of sensation.

(*b*) I look with considerable doubt on the reports of Brown-Séquard paralysis observed many years ago in which the statement is made that there was total loss of tactile sensation in the contralateral side, the lesion being strictly confined to a hemisection in extent. In the view of more recent observations it seems to me most likely that the extent of the lesion has been incorrectly judged.

(*c*) The cases where there was undoubtedly tactile anesthesia of the contralateral side of greater or less profundity are explained either by the fact that the knife or the pathologic process has invaded the posterior column of the opposite side or else that the wound has caused such an outpouring of serum or such a zone of inflammatory change that the centripetal impulses are impeded in the opposite posterior column.

The reason that Mann's hypothesis does not here obtain is that the antero-lateral ascending column is cut and there is no possible pathway for contralateral tactile stimuli.

(*d*) It may be advanced that there is an incongruity in my argument in that if the afferent pathway for tactile sensation of the opposite posterior column be invaded, why is not also the pathway for muscular sense disturbed? As a matter of fact one never sees loss of muscular sense on the contralateral side of the body.

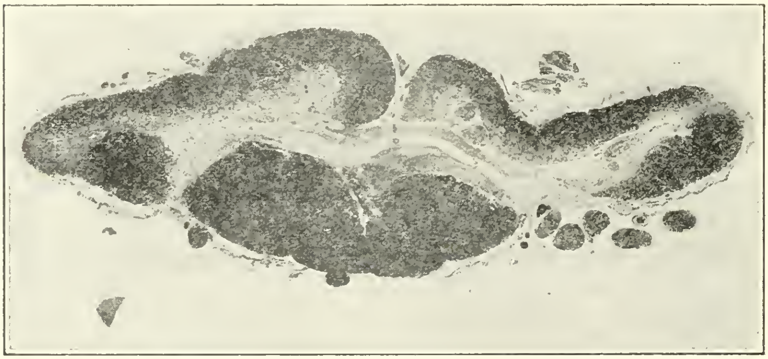

FIG. 2. First Thoracic Segment. Weigert Preparation.

The reason is that the fibers for tactile sensation are in all probability the long axones which pass as soon as possible toward the posterior median septum. I realize that this goes against the teaching of von Bechterew, but I have failed to see any justification for von Bechterew's views in the work he has done. The muscular sense fibers, on the other hand, probably enter the gray matter of the posterior horn without passing much more medialward than Wernicke's root entrance zone. In other words the fibers subserving muscular sensibility are as far separated as they well can be in the posterior columns.

Maillard, Lyon-Caen and Moyrand have recently reported (10) a case of Brown-Séquard paralysis which well repays careful study. In this case there was a stab-wound involving the anterior part of the right side of the spinal cord between the third and fourth anterior cervical roots. The lesion had destroyed all of the antero-lateral ascending tract, the antero-lateral ground bundle, the most anterior part of the direct cerebellar tract, the anterior half of the crossed pyramidal tract and the most anterior fibers of the posterior columns.

The interesting points in symptomatology were as follows: there was complete *flaccid* paralysis of the right arm and right leg, and concerning the tendon reflexes "sont extrêmement diminués à droit et il faut les recherches avec soin pour obtenir un faible mouvement." We know that when in cases of fracture dislocation the trauma to the cord is the equivalent of a complete section, the paralysis is of a flaccid type with loss of reflexes, but here the injury has not cut more than half of the pyramidal tract. The authors attribute this to "choc traumatique." There was complete preservation of tactile sensibility. Evidently the authors consider that the pathway for tactile sensation lies in the posterior columns, as they say "pas de trouble de la sensibilité tactile puisque le cordon postérieur est intact sur presque toute sa surface, . . ."

Through the kindness of Dr. Charles W. Burr I was enabled to study the following case during life, and after death to examine the brain and spinal cord.

J. B., male, aged 66 years, white, was three times an inmate of the Philadelphia Hospital, where he died, March 16, 1908.

The clinical diagnosis was syringomyelia.

History.—He denied syphilis, but had had gonorrhea once. He had used alcohol in moderation. At 26 years of age he had an attack which he called rheumatism and which left him with a deformity of his left wrist, having the appearance of a luxation of the lower end of the ulnar. This was possibly an arthropathy.

In 1885 (when 43 years of age) he began to get weak and in four or five weeks was unable to walk. The inability to walk has not been a constant symptom. At times there has been dribbling of urine. There was marked skoliosis with the concavity to the left in the thoracic region and to the right in the lumbar region. He was not able to extend the legs beyond a right angle, at which position they became fixed.

The biceps and triceps jerks were absent on each side. The patellar tendon and Achilles tendon jerks were not pronounced on either side, possibly owing to contractures. The Babinski reflex was present on each side. Scars from burns were present on all four extremities.

There was *analgesia* and *thermo-anesthesia* of all four extremities and trunk up to the mandible and extending above this border for a short distance immediately in front of the ears. There were areas in the lower limbs where pain and temperature were possibly correctly distinguished.

Sections were made from each segment of the spinal cord from the motor decussation of the medulla oblongata down to and including the second lumbar segment. Below this sections were made at intervals of one centimeter to a point four centimeters below the second lumbar segment. These sections were stained by the Weigert hematoxylin, hemalum-acid fuchsin, von Lenhossék's thionin phénique, hematoxylin-eosin, Weigert's elastica, van Gieson's and Marchi's methods.

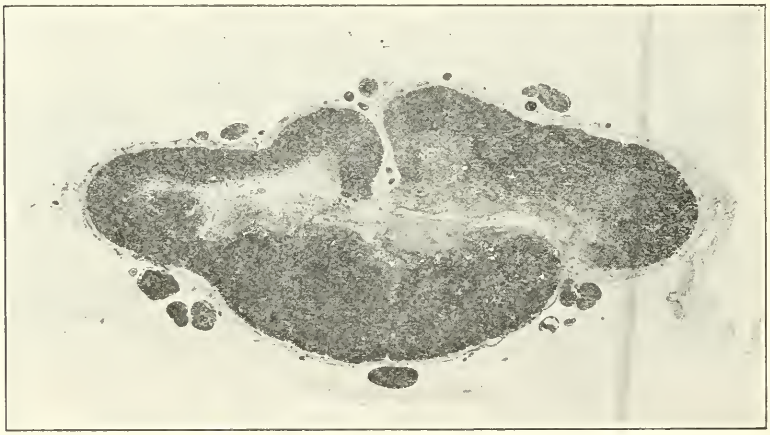

FIG. 3. Ninth Thoracic Segment. Weigert Preparation.

The point particularly noted is the destruction of the posterior horns, especially on the right side. The cavity or gliomatous process renders it impossible for fibers of the posterior roots to gain the position of the perykaria of the secondary sensory system. Furthermore, in most segments the destruction of the gray matter of the posterior horns is so extensive that these parykaria themselves have been destroyed.

This condition of affairs is present down to and including the eleventh thoracic segment. Below this point the gray matter of the cord is free from gliomatous or cavity formation sufficient in extent to cause interruption of sensory fiber tracts. And yet this patient had an almost complete syringomyelic dissociation of sensibility in the lower limbs. It becomes an interesting anatomico-physiological problem to explain where the break in the temperature and pain pathways for the lower limbs has occurred.

In view of the above finding it becomes plausible that the entering posterior root fibers of the lumbo-sacral segments subserving pain and temperature sense ascend for a considerable distance in either the posterior column or the posterior horn of the same side before making synapse with the secondary system of neurons whose axis cylinders cross in the anterior white commissure. This would account for the syringomyelic dissociation of sensibility in the lower limbs when the lumbo-sacral segments presented such a normal histological picture.

There is a condition found in the lumbo-sacral region which has caused me some speculation. Extending downward for a distance of about four centimeters below the first lumbar segment there is considerable amount of albuminous exudate about the anterior central arteries as they enter the substance of the cord at the bottom of the anterior fissure. The extent posteriorly of this area of exudation is considerable, reaching in places the plug of cells marking the position of the central canal. The longitudinal extent of this process is so great that I am not sure that it could not interfere with the function of the anterior white commissure. These central arteries are very close together, being about 0.125 apart. I found by using the Marchi method in these segments distinct degeneration of the anterior white commissure.

On recapitulation we may note here a case in which the pathologic process has completely isolated the posterior columns from the rest of the spinal cord from the first cervical to the eleventh thoracic segments, inclusive. There is perfect preservation of tactile sensibility and loss of pain and temperature sensibility. There is very diagrammatically demonstrated the lower limit of the trigeminal area. The cavity formation in the gliomatous tissue is most marked on the right side.

The case demonstrates: (1) the pathway for tactile sensations lies in the posterior columns; (2) with total cutting off of all the afferent pathways of the spinal cord with the exception of the posterior columns tactile sensation is not in the least impaired; (3) the validity of Mann's hypothesis is in no way questioned by the findings of this case.

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CENTRAL PAIN. A PATHOLOGICAL STUDY OF EIGHT CASES[1]

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An opportunity to study seven cases of hemiplegia with painful manifestations was afforded through the courtesy of Dr. Wm. G. Spiller, whom it gives me great pleasure to thank for the material of these cases. My own case of hemiplegia makes eight in all, in which the paralyzed limbs were the seat of pain, spontaneous in some and manifesting itself when pressure or movement of the affected limbs was made in others. While these cases more or less confirm the opinion of von Monakow, that the pathology of central pain is yet uncertain, it was possible to draw some important conclusions from a study of a comparatively large number of new cases, only twenty-four cases having been found in the literature of the subject after a careful search.

Case 1. R. F., age 74, presented negative family and previous histories. Two months before admission to the Philadelphia Home for Incurables, on October 4, 1904, without premonitory symptoms there was sudden right-sided hemiplegia involving the face, arm and leg, associated with disturbance of the speech. Upon admission to the Home the disturbance of speech and the facial palsy had cleared up almost entirely.

Upon examination there was partial paralysis of the right arm and leg and slight spasticity in the elbow, shoulder and knee joints. The knee jerk was exaggerated. There was no gross sensory change.

The patient constantly complained of pain on the right side of the body, including the arm and leg. Three weeks before death, she had a second apoplectic attack resulting in spasticity on the left side of the body as well as an increased stiffness on the right side. The knee jerks were increased on both sides and equal. There were ankle clonus and Babinski phenomenon present on the right but not on the left.

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[1] Read by title at the Thirty-eighth Annual Meeting of the American Neurological Association, May 30, 31, and June 1. From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania, and the Philadelphia Home for Incurables.

No gross lesion was found at the autopsy. The basal ganglia on transverse section presented a cribriform appearance on both sides, and sections from the right optic thalamus and the left lenticular nucleus were stained with hemalum and acid fuchsin and by the Weigert method.

A study of these sections showed a space formation, a rarification of the tissue around the blood vessels and some perivascular round cell infiltration. These space formations were very numerous. In the pons there were scattered throughout the whole transverse section, foci consisting of thickening of the glia. At these places the tissue stains poorly with the Weigert stain. These diseased areas were present in the motor tracts on both sides and in the left lemniscus.

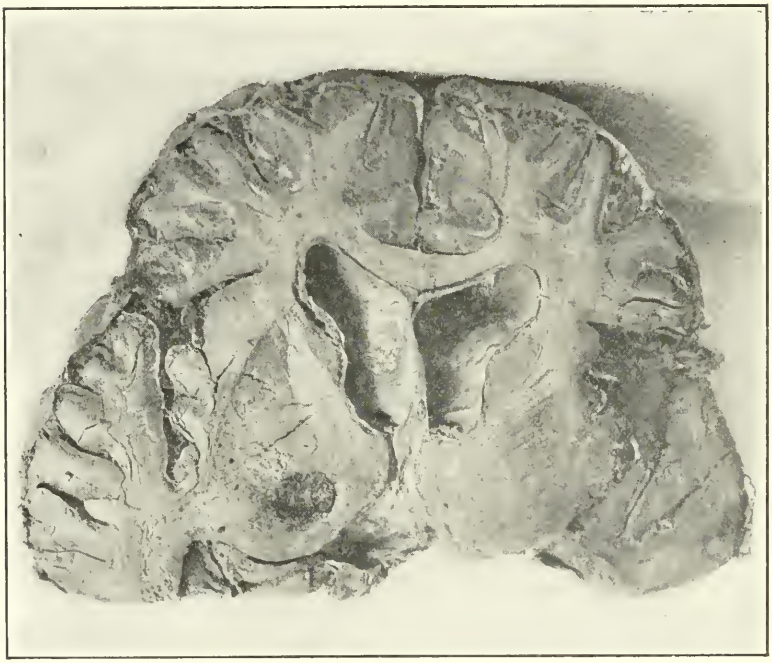

Fig. 1. Case 2. Hemorrhage into the Left Optic Thalamus Implicating Slightly the Internal Capsule.

The left pyramid in the medulla oblongata was moderately degenerated.

In the cord from the cervical to the lumbar region, there was marked degeneration of the right crossed pyramidal tract.

Case 2. F. S., a man of 65 years of age, an engineer, was admitted to the Philadelphia General Hospital January 24, 1900. A month previous to admission he had had an apoplectic stroke.

resulting in paralysis of the right arm, leg and face. The patient was apparently very deaf in the right ear. He seemed to understand questions, but could not give intelligent replies. There was no atrophy or other atrophic changes in the right arm. Sensation

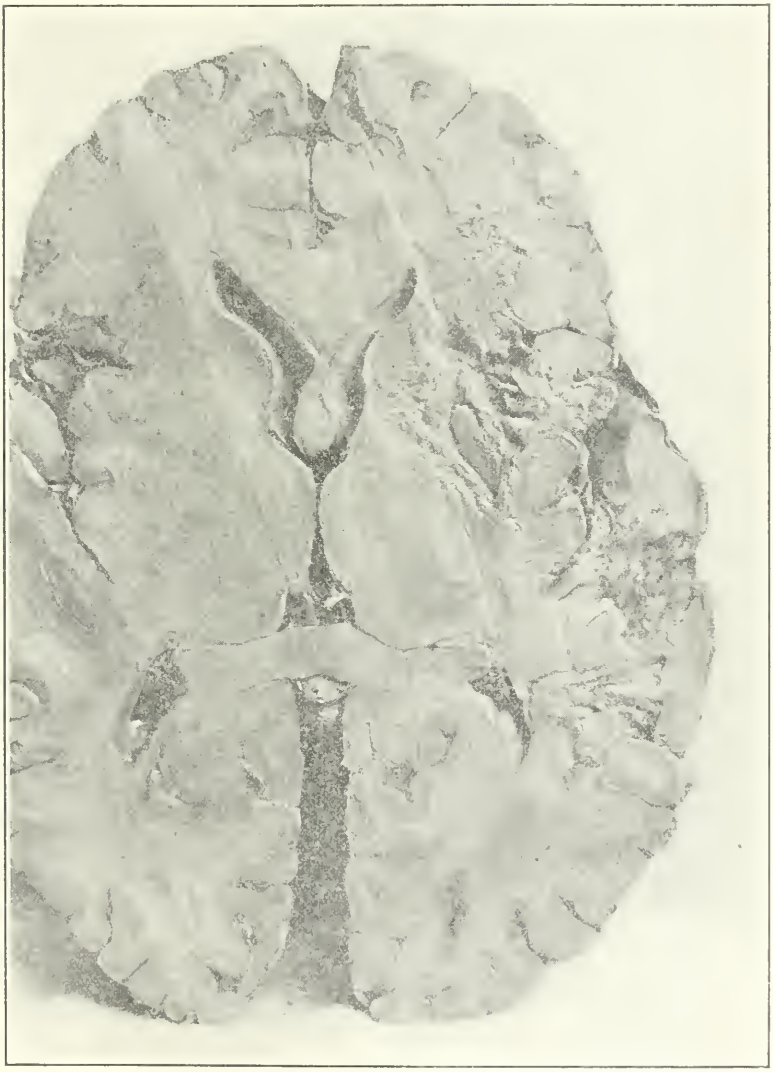

FIG. 2. CASE 3. Extensive Area of Softening in the Right Lenticular Nucleus Implicating the Rolandic Cortex.

apparently was intact and the patient partly withdrew the arm on deep pressure by pain pricks with expressions of pain. Sensation in the legs was apparently normal.

Intense pain in the right arm on pressure or movement was complained of.

A large hemorrhage measuring three quarters by one half inch was found in the left optic thalamus at the level where the third division of the caudate nucleus appears well defined on transverse section. This hemorrhage extended somewhat into the centrum ovale and implicated in part the internal capsule.

CASE 3. S. M., man of 54, was admitted to the Philadelphia General Hospital January 1, 1910, and died February 16, 1910. There was tuberculosis in the family history, but otherwise the family and previous histories were negative, syphilitic infection being denied.

Three years previously he suffered a stroke of apoplexy, resulting in paralysis of the left side, from which he recovered entirely. Two weeks before admission to the hospital he suffered a second apoplectic attack, followed by paralysis of the left arm and leg. Touch and pain senses were not impaired, but temperature sense was impaired in both arm and leg. Sensation on the right side was normal for both pain and temperature. The left side of the face in the lower portion was paralyzed. Passive movements of the left leg produced great pain and he expressed great fear of having the left arm moved. All of the reflexes were increased on both sides. There was no ankle clonus on either side.

The patient died of pneumonia six weeks after admission.

There was an extensive area of softening in the right lenticular nucleus, extending outward and at this level implicating also in part the cortex anterior to the fissure of Rolando.

Microscopic examination of the paracentral regions showed a marked round cell infiltration of the pia on the left side. On the right side there was some perivascular round cell infiltration of the cortex. The intima of the middle cerebral artery was markedly thickened. Examination of the medulla oblongata and spinal cord showed no change when studied by the Weigert stain, but the pyramidal tract on one side showed degeneration by the Marchi stain.

CASE 4. U. F., man of unknown age, admitted to the Philadelphia Hospital April 19, 1909, presented a negative family history. There is nothing in his previous history bearing on the case, except that he was a heavy drinker. Eight years ago, while working in a room at a high temperature, he suddenly became unconscious, and afterwards there was paralysis of his right arm and leg and inability to speak.

On examination the right arm was contracted at the elbow and at the wrist, and the fingers were flexed on the palm. The right leg was held in the extended position and was spastic. The left

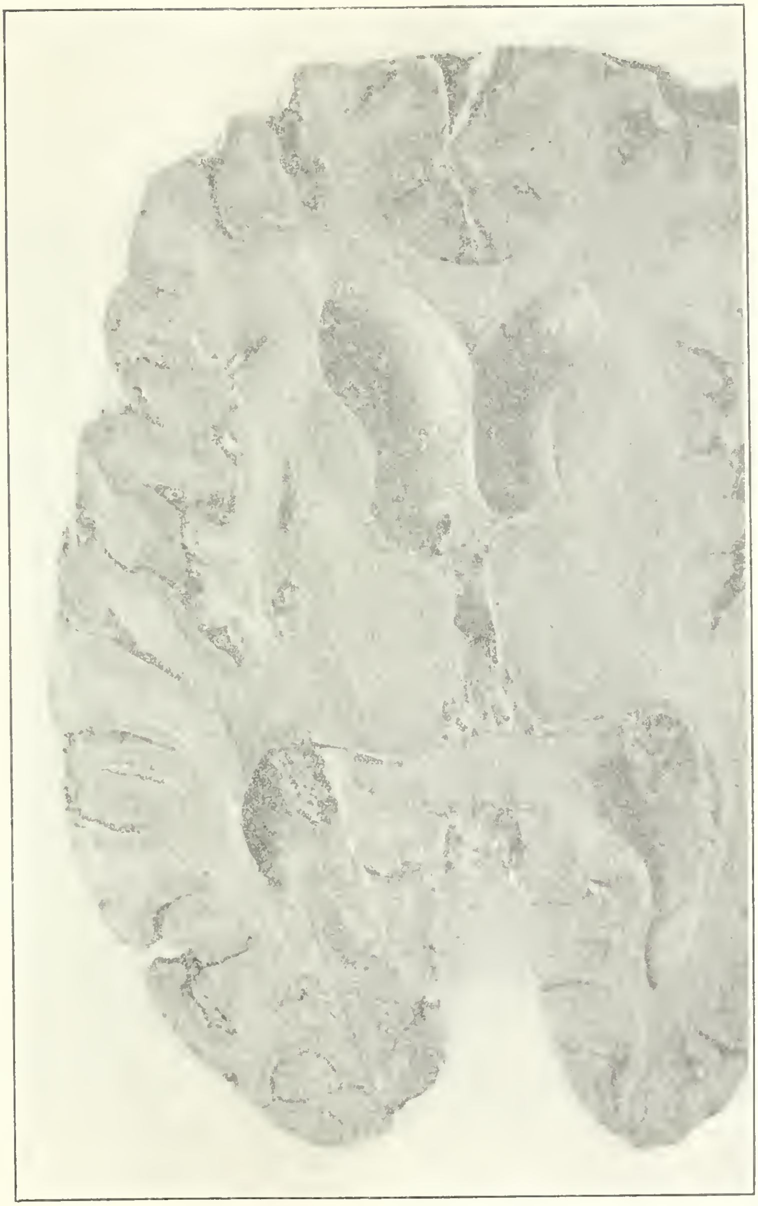

Fig. 3. Case 4. An Old Cyst in the Left Lenticular Nucleus Involving the Knee of the Internal Capsule.

arm and leg appeared to be normal. The knee jerks were increased on both sides but more so on the right, where there were also an ankle clonus and Babinski phenomenon. Sensation to touch and pain was normal.

The patient complained of sharp pain and also jerking sensations in the right leg.

At the autopsy there was found an old cyst in the uppermost part of the left lenticular nucleus, involving the knee of the internal capsule. There was a round cell infiltration of the pia of the cortex. The pia of the medulla oblongata was thickened and also the seat of round cell infiltration. The right pyramid was degenerated.

In the spinal cord the left crossed pyramidal tract was degenerated as far as the lumbar region. In the upper thoracic region extending to the mid-thoracic region the direct cerebellar tract was slightly degenerated on the left side. In the mid-thoracic region the pia was infiltrated with round cells. The white matter in the anterior part of the cord showed a diffuse round cell infiltration and some vascular change. In the pia in this region there was a marked round cell infiltration. In the anterior horns there was also some perivascular round cell infiltration.

In the cervical region there was a sclerotic area in the anterior portion of the cord on the left side. There was some degeneration also of the posterior roots and a round cell infiltration about the roots. The condition was evidently specific and in the nature of a diffuse meningo-myelitis with involvement of the posterior roots.

Case 5. M. R., age 53, was admitted April 1, 1908, to the Philadelphia Hospital, with a history of having suffered from an apoplectic attack a few days previously. Upon admission he was completely aphasic and was unable to give any history. He showed by signs that he understood what was being said to him but was unable to articulate. There was no paralysis of the ocular muscles and he wrinkled his forehead equally well on both sides. There was some paresis of the lower half of the face on the right side and complete paralysis of the right arm and leg. The right arm was somewhat spastic, but the right leg was flaccid.

The left leg was slightly spastic. There was no paralysis of the left arm and leg. Babinski phenomenon was present on both sides. It was not possible to obtain a knee jerk on the right side and on the left it was diminished. There was incontinence of both feces and urine. As far as could be determined there was no impairment of touch, pain or temperature sense. There was some apraxia present. When he was requested to touch his nose he touched his chin or when he was asked to close the right eye he closed the left. There was no hemianopsia.

Movements of the right arm appeared to be somewhat painful and pressure over the calf muscles appeared to give a great deal of pain.

A recent hemorrhage was observed in the left cornu ammonis. There was also a linear cyst in the left external capsule.

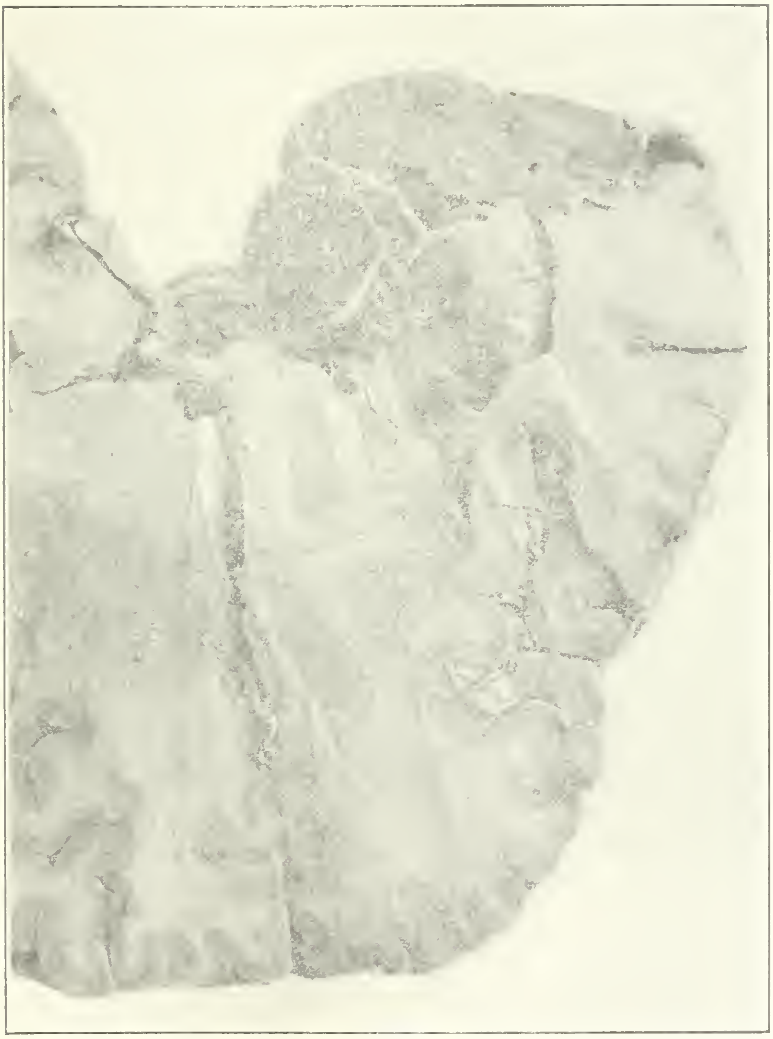

Fig. 4. Case 5. Recent Hemorrhage in the Left Cornu Ammonis and a Linear Scar in the Left External Capsule.

A microscopic study of the spinal cord showed that the crossed pyramidal tract was a little pale by the Weigert stain on one side and one of the pyramids of the medulla oblongata showed slight degeneration.

Case 6. M. G., age 68, was admitted to the Philadelphia General Hospital May 1, 1904. The family and previous histories are negative, except that twenty years previous she was seized with an apoplectic attack, which left her paralyzed on the left side. She was admitted to the hospital for contusion of the left knee.

She suffered from a second stroke five years ago, presumably on the left side, though the history does not state definitely. Since this time she has suffered from sharp shooting pains in the left arm and leg, but more so in the arm.

On examination there was diminished power in the left arm. The left hand and forearm were markedly atrophied, especially in the hand. The biceps, triceps and wrist jerks were prompter than normal. Touch and pain senses were preserved. She was unable to move the left lower leg, except at the ankle and toe joints. The knee jerk on the left could not be obtained. Ankle clonus and Babinski phenomenon were present on the left.

Sensation for touch and pain was preserved, and it was stated that she could feel more distinctly on the left side of the body than on the right. Power in the right arm and leg was preserved.

She not only complained of spontaneous pain in the left arm and leg, but there was tenderness to pressure over the bony parts in these localities, though present also over the soft parts.

On July 28, 1904, a flap was made from the median nerve and inserted into a longitudinal incision in the sheath of the musculospiral nerve. Some improvement in the power of extension in the fingers and wrist was noted. On November 16, 1907, she suffered from a clonic convulsion, mainly limited to the right arm and leg, and died a few days later.

There was present at the autopsy an old scar in the right optic thalamus, extending into the internal capsule. There was also a small hemorrhage beneath the floor of the descending horn of the lateral ventricle at about the junction of the occipital and temporal lobes.

In the medulla oblongata one of the pyramids showed degeneration by the Weigert stain.

In the cervical region of the spinal cord, the right direct pyramidal tract and the left cross pyramidal tract were degenerated. This degeneration did not extend as far as the lumbar region.

Case 7. G. S., age 69, was admitted September 16, 1904, to Philadelphia General Hospital and died March 28, 1905. Family and previous histories were negative.

For two years previous to admission he had been subject to attacks of vertigo and had been a heavy drinker for years. On the day of admission he had an attack of vertigo and fell, since which time he had been unable to walk.

On admission there was slight paralysis in the lower portion of right side of face. Motion and resistance to passive movements were good in the right arm and leg. The left arm was contractured and spastic and the grasp was very poor. Sensation to touch and pain was better on the right side than on the left. Motion and resistance to passive movements were poor in the left leg. Both knee jerks were exaggerated, and ankle clonus and Babinski phenomenon were present on both sides. Sensation to touch and pain was diminished on the left side. The left leg was spastic and the bladder sphincter was incontinent.

He complained of sharp shooting pains in all his limbs, especially in the legs. There was also pain on pressure in all the limbs over the nerve trunks, especially over the legs.

On December 22, 1904, he suddenly noticed that he could not use his right hand, which upon examination was found to be paretic. There was noted a week later some difficulty in phonation. There was no loss of sensation on the right side.

No gross lesion was found at the autopsy. The pia of the paracentral, frontal and occipital regions, as well as the pia of the pons, medulla oblongata, and spinal cord, was slightly infiltrated with round cells and the blood vessels were thickened.

In the cortex there was some perivascular round cell infiltration.

There was an area of softening in the pyramidal tract on one side of the pons, causing descending degeneration.

This case was reported in a paper by myself, published in the *American Journal of the Medical Sciences*, May, 1906.

Case 8. E. J., was admitted to the Philadelphia General Hospital on June 12, 1903. There was no history of paralysis or nervous trouble in the family and he claimed to have had no disease previous to the present trouble, denying specific history.

Several weeks before admission he suddenly became unconscious. Upon regaining consciousness the right arm and leg were paralyzed and he was unable to speak distinctly.

On examination the right arm and leg were entirely paralyzed. He moved his left arm and leg but with some apparent loss of power. Sensation for touch and pain was lost and the temperature sense impaired on the right side of the body, extending one inch to the left of the median line and including the right arm and leg.

Hemianopsia was absent. The knee jerks were increased very much on both sides, but more so on the left side. There was a Babinski phenomenon on the right but no ankle clonus. There was apparently some motor aphasia.

The patient complained of great pain in the right arm and leg when moved passively, though they could be touched or pressed with impunity and no pain was complained of when the limbs were at rest.

There was a hemorrhagic scar in the left lenticular nucleus and one in the external capsule. There was also sclerosis of the cerebellar lobe.

The middle portion of the foot of the peduncle on one side was markedly degenerated.

Summary.—In four of these cases the pain was spontaneous (cases 1, 4, 6, 7). In case 1, there was no pain to pressure, the pain being entirely independent of pressure or movement of the limbs. The multiplicity of the lesion in this case makes it impossible to draw any definite conclusion, though it must be remembered that the basal ganglia were the seat of a widespread lesion of a minute character.

In case 4, the pain was also spontaneous and there was no note of pain on pressure. The lesion was in the lenticular nucleus.

In case 6, the pain was not only spontaneous, but when the bony parts were pressed upon there was also complaint of pain. There was an old scar in the contralateral optic thalamus.

In case 7, sharp shooting pains were complained of as well as pain on pressure over the nerve trunks. In this case the lesion was one of cerebrospinal lues, and the meningeal irritation at the roots may have been the cause of the pain.

In four of the cases the pain was only manifested upon movements of the limbs or pressure.

In case 2, there was pain in the right arm on pressure or movement. The left optic thalamus was the seat of a hemorrhage, which implicated also the internal capsule.

In case 3, passive movements of the left leg gave great pain. The lenticular nucleus was the seat of an area of softening which involved also the motor cortex.

In case 5, pressure over the calf muscles and the right arm was painful. A recent hemorrhage was seen in the left cornu ammonis and a linear scar was present in the left external capsule. Some degeneration of the pyramidal tract on one side could be seen by the Marchi method.

In case 8, passive movements of the right arm and leg gave pain, though pressure was painless when the limbs were at rest. The lesion was in the left lenticular nucleus and in the external capsule.

The painful conditions found in these cases were independent of contracture. The possibility of the presence of neuritis as an explanation of the pain must be remembered, though in these cases above cited it was not suspected.

An analysis of these cases makes it apparent that there is no one pathological lesion to explain pain of central origin.

In three of the cases (cases 8, 4, 3) the lesion was in the lenticular nucleus; in two of the cases (cases 2, 6) an old hemorrhage was found in the optic thalamus; in one case (case 7) there was a widespread specific lesion, causing numerous foci of degeneration; in one (case 1) the optic thalamus and lenticular nucleus as well as the pons were the seat of an arterial lesion causing small foci of degeneration; and finally in one case (case 5) the cornu ammonis and the external capsule were the seat of a hemorrhage.

In case 4, the presence of a diffuse myelitis and meningeal infiltration associated with involvement of the lenticular nucleus excludes the possibility of attributing the pain to lesions of the white matter of the brain alone. The meningeal and root implication could readily explain the sharp pain and jerking sensations in the leg. The case is useful, however, in this study as illustrating what I take to be not an infrequent cause of pain in hemiplegia, namely syphilis, the lesion causing the palsy originating in the same process which gives rise to the round cell infiltration.

In case 7, there was a widespread arterial change of specific origin, giving rise to small foci of softening and causing the double hemiplegia. There was round cell infiltration of the pia of the cord though moderate, which may not have been sufficient in itself to cause enough irritation of the roots to give rise to the pain in the leg, but it is perhaps justifiable to assume that some undiscovered foci of softening was present in the sensory tracts and that thus the pain could be explained. It is assumed that irritation of the sensory tracts may cause pain referred to the periphery.

There was no other sensory disturbance in five of the cases (1, 2, 4, 5, 6). In two of the cases, however, there was loss of sensation to pain and touch (7, 8); and in one of the cases a loss of temperature sense (3).

Central pain described by Oppenheim (1) as analgesia dolorosa when anesthetic areas are the seat of spontaneous pains, according to this observer, is of frequent occurrence. At the same time the observations in literature with autopsy, where pain has been of central origin, are not very numerous. While the observations of Morot (2) and Duchek (3), reported respectively in 1785 and

1864, show that this condition has been recognized for a long time. the first analysis from a pathological standpoint of the cause of central pain was made by Edinger (4) in 1891. Previous to this date, however, several cases had been reported.

Lauenstein (5) in 1887 reported a case of athetosis, associated with severe pain in the hand and forearm which were intensely red and felt cool. There was a fresh hemorrhage in the right optic thalamus the size of a bean.

In 1883 Greiff reported a case of post-hemiplegic chorea, residual dementia and left-sided paresis in which there was burning pain in the left arm and leg, which were hyperesthetic and painful to pressure. There was a focus in the right optic thalamus, one at the base of the right occipital lobe, one in the cerebellum, and a small focus in the left upper part of the pons, the last, however, in his opinion not causing any symptoms.

Reichenberg (7) in 1897 described a case of paralysis of the left side of the body, hemianopsia and severe pains beginning four days after the onset of the palsy. The lower part of the arm, the trunk and the left leg were partially anesthetic and there was also some hyperesthesia of the face and left upper arm. Softening in the right hemisphere was found, involving a large part of the lower parietal lobe and implicating also the posterior part of the internal capsule and the optic radiations in their dorsal part.

Henschen (8) in 1890 described a case of pain in the right arm followed by palsy and later by choreiform movements of the thumb and associated with hemianopsia. There was a small hemorrhage in the optic thalamus in the vicinity of the internal capsule and optic tracts. Since Edinger's paper a few observations have been made in the literature on the subject, namely, the cases of Oppenheim and Koehler (9), Handford (10), Biernacki (11), Schaffer (12), Touche (13), Zawadsky and Bregman (14), Leyden (15), Economo (16), and Holmes and Head (17). To these may be added those cases described by Roussy under the name of the thalamic syndrome, consisting of hemianesthesia to all forms of sensation, hemiataxia, slight hemiplegia, hemianopsia, choreiform movements, and severe pains in the paralyzed limbs. Cases of this sort have been described by Dide and Durocher (18); Dejerine and Roussy (19); Roque, Chalier and Cordier (20); Long (21), and Paillard and Lelievre (22).

In Dide and Durocher's case, the lesion was in the caudate nucleus and in the superior third of the thalamus, both of recent date, and there were two older lesions in the thalamus, bordering on the posterior part of the internal capsule.

The lesions in the three cases of Dejerine and Roussy permitted them to conclude that the characteristic seat of the focus in the thalamus syndrome was in the external nucleus and in a situation which encroached upon the internal and middle nuclei of the thalamus, and which only invaded a part of the posterior segment of the internal capsule.

In the case of Roque, Chalier and Cordier there was a hemorrhage into the posterior part of the right optic thalamus. In one of Long's cases there was a focus in the retro-lenticular segment of the internal capsule, and one in the marginal gyrus; in the second case a lesion occupied the external nucleus of the thalamus and the pulvinar, besides which there were numerous foci in the occipital lobes, the central part of the left hemisphere, the pons, and in the medulla oblongata.

In Paillard and Lelievre's case there was a hemorrhage in the retrolenticular region invading the posterior part of the internal capsule. The posterior and external part of the optic thalamus was also invaded.

These cases can be grouped pathologically as follows: (1) Lesions of the optic thalamus (Dide and Durocher; Roque, Chalier and Cordier; Greiff; Lauenstein; Biernacki; Long; Eisenlohr; Edinger; Henschen; Holmes and Head; Paillard and Lelievre); (2) lesions of the caudate and lenticular nuclei (Zawadski and Bregman); (3) lesions of the pons (Morot, Duchek, Economo); (4) lesions of the parietal regions (Oppenheim and Koehler, Reichenberg); (5) lesions of the frontal convolutions (Touche, Handford); (6) widespread degeneration involving the lemniscus (Schaffer); (7) lesions of the medulla oblongata (Mann's (23) clinical case).

Pain in disease of the nervous system, excluding affection of the nerves, has been looked upon as originating in irritation of the meninges or of the spinal roots.

Generally speaking the consensus of opinion has been against the view that implication of the central nervous tissue when irritated can cause painful manifestations.

At the same time it has been assumed by certain authorities (Zawadski and Bregman, Reichenberg, Nothnagel (24)) that central pain may be due to irritation of the sensory fibers in the internal capsule.

Just what the mechanism of the production of central pain is has been a matter of considerable dispute.

Lewandowski (25) did not believe that it was certain that peripheral irritation could be excluded in these cases or that the pain was simply referred peripherally. Goldscheider (26) on the contrary looked upon these cases as resulting less from irritation of the white matter of the central nervous system than from implication of the gray nuclei. Anton (27) was not sure what fibers were implicated in central pain, but looked upon the presence of subjective sensations and paresthesia occurring in lesions of the parietal cortex as suggestive in localizing painful sensations (see Handford's case).

Economo believed that there were fibers in the lateral part of the lemniscus which conveyed not only pain sensations to the skin but to the bones and muscles, and that these fibers are in the "spino-tektalen," as well as in the "spino-thalamic" tracts. He believed that injury to these pain tracts (Schmerzbahnen) was the cause of central pain.

The lesion may be in the corona radiata at the junction of the ascending frontal and second frontal convolution (Touche), or in the frontal region (Ballet (28)), or in the right parietal region (Oppenheim, and Koehler, and Reichenberg), or in the paracentral region (Handford).

According to von Monakow (29) pain occurs in foci situated in the paracentral region or in irritating lesions in the posterior segment of the optic thalamus. Anesthesia dolorosa, he states, is caused by lesions of the pons. He admits, however, that the anatomical substratum for conditions of sensory "Reizerscheinungen" is not yet certain.

In this connection it is interesting to note the association of pain with organic hemichorea and athetosis as in the cases of Touche, Henschen, Greiff, Lauenstein and Edinger.

While there is no doubt that lesions of the parietal and motor cortex, as well as of the pons and medulla oblongata (Mann), may cause central pain, the chief seat of lesions appears to be the basal ganglia and mainly the optic thalamus. The cases of Greiff, Henschen, Holmes, Edinger, Biernacki and Eisenlohr were all instances of this kind, as well as those cases of the thalamic syndrome above cited.

On the other hand it may occur when the optic thalamus is intact as in my own cases and that of Zawadski and Bregman, in which the lenticular nucleus was involved by a lesion implicating at the same time the internal capsule. It may be the result of a widespread lesion, as in the case of Schaffer, where the focus involved the pulvinar, the left lingualis and fusiform lobes, the cuneus, the corpora quadrigemina and the cornu ammonis, as well as the "Schleifenschicht." Secondary degeneration of the "Längsbundle," part of the splenium, contralateral tapetum and descending degeneration of the pyramidal tracts from a focus of softening in the right pyramid of the medulla oblongata were also present. He concluded that the lemniscus is related to central pain.

Though there is little in the study of the lesions in these cases cited, as well as in the cases reported in this paper, to suggest an entirely satisfactory explanation for central pain in all the cases, it seems not improbable that there are separate tracts for pain as there are tracts for other forms of sensation.

Where these are located is a matter of conjecture. It is significant that lesions in the motor regions (Hoppe's (30) cases) and in the parietal region gives rise to parasthetic and painful manifestations, also that in some forms of hemiplegia parasthesia may exist without loss of sensation.

That pain fibers are separated from tracts transmitting other forms of sensation seems further probable when we note that in my cases no other form of sensory disturbance occurred in five instances, while the loss of sensation of pain and touch occurred only in two cases, the lesions found being lues in one and scars in the left lenticular nucleus and the external capsule in the other. In one other case the temperature sense was lost, the right lenticular nucleus and the brain substance of the Rolandic cortex being involved. The lack of uniformity of the lesion makes it impossible in these cases to draw any conclusions as to the location of pain tracts, granting their existence.

In this connection the conclusions of Head and Holmes (31) are interesting. They believe that the "optic thalamus contains the terminations of all sensory paths; here the sensory impulses are grouped afresh and redistributed in two directions, on the one hand to the cortex, and on the other to the gray matter of the optic thalamus itself. It contains a mass of gray matter, the essential organ of the thalamus, which forms the center for certain fundamental elements of sensation. It is complementary to the sensory cortex, and exercises different functions in the production of sensation. The lateral part of the optic thalamus is the organ through which the cortex can influence the essential thalamic center, controlling and checking its activity. The excessive response to affective stimuli, so prominent a feature of lesions in this situation, is not due to irritation but to removal of cortical control."

It seems very probable that the optic thalamus is a great sensory receiving and distributing center. Pain fibers no doubt are connected in some way with these centers, but a study of my cases would lead me to believe that pain fibers exist also in the nerve tracts between the cortex and optic thalamus, and possibly also in connection with the Rolandic cortex as well as other regions of the cerebrum.

Another element may be considered in the discussion of these cases. The cause of the pain may not be directly due to the principal lesion described. In most of the cases there is also an arterial sclerosis which it is reasonable to look upon as a possible etiological factor in causing irritation or disturbed nutrition in fibers whose physiological function is concerned with painful sensations.

Conclusions.—It seems justifiable to conclude that central pain may be found associated with lesions in many localities, but the cases in which the optic thalamus is implicated are in the majority. Serial sections should be made in all cases before definite conclusions may be drawn. This was not practical in my cases, as most of the material was preserved for teaching purposes as gross specimens.

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INTERMITTENT CLAUDICATION IN THE UPPER EXTREMITIES[1]

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Intermittent claudication or dysbasia intermittens angiosclerotica of the lower extremities has been described by Charcot, Sabourin, Magrez, Hunt, Riesman and many other writers, but very few cases have been reported (Nothnagel, Goldflam, Wwedenski, Embsden, Kronenberg and others) where the involvement was of the upper limbs.

In addition to this latter type several writers have reported affection of the tongue and face (Goldstein, Goldflam and others).

The following case which Dr. Spiller has kindly given me the opportunity of reporting, from his service at the University of Pennsylvania Dispensary, presents unusual symptoms, in the absence of any involvement of the lower limbs, in the almost continuous pain present, this extending even through the interparoxysmal periods, and in the absence of any abuse of the system that might, by a toxemia, cause arterial changes, but with an indirect cause apparently in frequent exposure to cold and overexertion, with the possibility of an acutely exciting cause in an attack of influenza.

Dysbasia intermittens angiosclerotica comes on, as a rule, in the years past middle life, during the period when weakness and a more or less sclerosed condition of the arteries would be expected. It occurs more frequently in the male sex, and when it does affect women, according to Wandel and Goldflam, the upper extremity type is the more common, this being attributed by them to the greater exposure and exertion of the arms as compared with the lower limbs, particularly in women of the working class.

In speaking of this tendency of the disease to involve the parts most used, it might be of interest to mention a condition occurring in marble cutters who use the pneumatic chisel. Through

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the kindness of Dr. Mills I was afforded the opportunity of questioning a patient under his care, who had a few of the symptoms caused by these automatic devices, and he informed me that the men who use them are frequently compelled to stop their work to rub their hands and wrists, because they become so numb and cold and white and painful, resembling greatly the picture presented by Raynaud's disease, and that they are often required to stop using the devices entirely, on account of the severity of the symptoms.

This is probably a condition caused by an occupation and similar to or identical with dysbasia angiosclerotica.

The exposure of the ankles to cold and wet is one possible reason for the great percentage of affection of the anterior and posterior tibial arteries, the anterior tibial and the ulnar (Bregmann) being the most frequently affected.

Some of the cases published during the last few years are well worthy of comment. Goldstein's case is of especial interest in that it brings up the possibility of there being a cerebral arterial spasm as the basis of cases such as he reports, wherein the leg, arm, eyes, larynx and speech were all involved, particularly as the motor symptoms were on the right side of the body. The general symptoms present in this case also point to an angioneurotic tendency, or at least to weakness in some degree, of the nervous system. Oppenheim believes inherent weakness of the nervous system to be of great importance as a predisposing factor in these cases.

Typical cases, with intermittent loss or diminution of muscular power, coldness and pallor of the affected limbs, pain and occasionally cramping, the absence of palpable pulsation in the radial or dorsal foot arteries, or even of the femorals, are all no doubt aided in their onset by any causes leading to arteriosclerosis.

Erb lays more stress upon the use or abuse of tobacco as a causative agent in arteriosclerosis than any other factor outside of lues, this latter being of course a well-known and most active cause.

Erb also reported cases of intermittent claudication with an acute onset resembling almost an infection and which he believed to have been due to an acute endarteritis brought on by severe exposure to cold and wet. Whether a like condition could occur in predisposed or partially diseased arteries, from an acute infection, such as influenza, as in my case, is not a certain point, but I think it deserves consideration.

As a rule pain is only present during the paroxysm (Kronenberg) and not when the patient is at rest and quiet and continuous pain, as in the case I am reporting, is very uncommon.

Many cases of so-called rheumatic muscular pains in the arms and legs are doubtless due, as Erb has suggested, to arterial changes, and might be considered as early stages of intermittent claudication. This pain and soreness of the muscles must not be confused with the symptoms in those cases in which the trouble is caused, according to Yawger and others, by nodular deposits in the muscular or intermuscular tissue and are such common causes of obscure as well as acute painful conditions.

Intermittent claudication may resemble in many respects a myasthenia, even showing the typical myasthenic electrical reaction, considered by many as pathognomonic of myasthenia gravis. Such a case was reported by Goldstein.

On the other hand, true myasthenia gravis may present an early symptom complex very difficult to distinguish from that of intermittent claudication, and I recall a case of this character, not yet fully recorded, which I saw at the University of Pennsylvania.

Venous changes or disease may give symptoms of claudication similar to those in cases of arterial disease, and a case of this kind has been described by Grieg and particular weight put upon the importance of looking for affection of the veins.

As so many of the cases reported have shown a distinct series of changes in the vessel walls, in many instances making it impossible to feel any pulsation, and as this hardening of the arteries has been suitably shown by radiographs, it demonstrates clearly that the arteriosclerosis is the basis of the disease. A secondary condition follows in the nerve trunks and fibers as would be expected where the loss in their nutrition is so great and in a case of Lapinsky's there was edema of the nerve sheath and marked changes in the endo- and perineurium, due to the excess of connective tissue.

Case W. D. (7075). Admitted to University of Pennsylvania Dispensary February 9, 1911. Man. Age 58. Slate quarryman.

Early history and family history negative. Patient smokes about 3½ ounces of tobacco per week. Has not used alcohol at all in last 25 years, and before then only in moderation. No venereal disease. About 15 years ago both bones of the left forearm were fractured and 10 years ago the left shoulder was partially dislocated by a fall from a small height. The man's occupation is such as requires continuous and rather severe or tiring use of both hands and the left hand has been particularly exposed to severe cold and wet.

Three weeks ago the patient had an attack of influenza and while confined to his bed during this illness he had a sudden onset of pain in the left upper arm, extending down the forearm, into the hand. The pain was very severe and the arm and hand became paralyzed so that no movement was possible in any of the muscles except those of the shoulder. The finger tips felt numb and the hand, forearm and lower part of upper arm became very white and cold. This attack lasted from two to two and a half hours and the symptoms are given in as near their order of occurrence as the patient could remember.

After this attack the patient could move all the muscles slightly but not so well as before the onset.

Remissions followed, four or five times daily for three days, but during these paroxysms the motor involvement was not so severe in character, becoming less marked in the successive attacks, so that at the present time the power in the muscles is fairly good though not normal.

Vasomotor phenomena no longer occur during the paroxysms.

Any prolonged effort causes intense pain in the left arm and forearm. This is not only present more or less constantly during the day, particularly when the hand is exposed to cold, but even in the night it is so excruciating as to keep the man awake for hours.

Examination: Right arm normal. Left arm slightly shorter than the right, due to the old fracture of the forearm. At this point of fracture there was a soft flabby mass of tissue anteriorly which may possibly be the pronator quadratus torn from its insertion and softened from disuse. All the muscles of the hand and forearm and the biceps and triceps were very weak. Reflexes in both arms about equal and slightly exaggerated—those of the legs normal. The brachial arteries in both arms were plainly palpable and not unduly sclerosed. Radial pulsation on the left was not obtainable and very weak on the right. Dorsalis pedis arteries not palpable on either side. Posterior tibial and femoral vessels on both sides easily distinguished though the latter were slightly weaker than normal. The heart, with exception of some accentuation of the second sound, was normal.

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THE DIFFERENTIAL DIAGNOSIS BETWEEN HYDROCEPHALUS, WITHOUT ENLARGEMENT OF THE HEAD, AND BRAIN TUMOUR BY MEANS OF THE X-RAYS.

By WILLIAM G. SPILLER, M.D.,

Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania; Corresponding Member of the Gesellschaft Deutscher Nervenärzte.

Occasionally the diagnosis between hydrocephalus and brain tumour is extremely difficult. In 1902 I reported a case, presenting this difficulty, that had been in the service of Dr C. K. Mills, and the clinical report was given in full at that time. It is sufficient to repeat here that the patient was a youth aged 19 years. He had had headache with vomiting occasionally for many years, but about six months before death the headaches became more severe, and walking began to be difficult, so that he staggered like a drunken person and had vertigo. Some exophthalmos was present. The patellar reflexes were exaggerated, but ankle clonus was not obtained. Slight patellar clonus was present on each side. Dr de Schweinitz found a well-marked beginning optic neuritis in each eye. Shortly before death the patient had slight tremor of the muscles of the arms and face, lasting a few seconds; this was followed by unconsciousness, stertorous breathing, and irregular and rapid pulse.

There was much in this case to suggest cerebellar tumour. The symptoms had been severe during a period of only about six months before death. The head was not unusually large and did not suggest hydrocephalus by its size.

At the necropsy the third and lateral ventricles of the brain were found much distended, the fourth was of normal size. The aqueduct of Sylvius was occluded by overgrowth of neurogliar tissue and ependymal cells.[1] The calvarium on the interior was deeply grooved by the convolutions of the brain, and at the sides was hardly thicker than a piece of paper. When it was held up to the light the atrophied portions corresponding to the convolutions allowed considerable light to pass through.

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[1] Spiller, *The American Journal of the Medical Sciences*, July 1902, Case 2.

At the time this report was made there appeared to have been no definite means of determining the correct diagnosis during the life of the patient. The symptoms had not persisted long enough to suggest the probability of hydrocephalus, and the skull had not yielded to the internal pressure in such a way as to cause a separation of the sutures, as the hydrocephalus developed after firm union of the bones of the skull had occurred. It was because of this firm union that the atrophy of the interior of the calvarium was so great. Whether the entire skull was atrophied or not, I cannot say; presumably it was. Some recent results obtained by Dr C. L. Leonard with the X-rays have made it desirable to revert to the above case.

At the March 1910 meeting of the Philadelphia Neurological Society, Dr Alfred Gordon presented a case, under the title, "A Case for Diagnosis," which to me appeared like one of hydrocephalus, although Dr Gordon considered this diagnosis uncertain. Dr Leonard[1] had made X-ray photographs of the head, which showed distinct areas of depression in the inner table of the skull. Having in mind the condition of the calvarium described by me in 1902, and recognising that a skull of this character would give just such pictures with the X-rays as those exhibited by Dr Leonard,[2] I expressed the opinion that Dr Leonard had obtained photographs showing atrophy of bone produced by pressure of the cerebral convolutions, and corresponding in form to these convolutions, and had made a valuable contribution to neurology. He had given us a means whereby we might be able to distinguish between certain cases of hydrocephalus and tumour during the life of the patient. It seems to be possible by means of the X-rays to prevent unsuitable operation in some of these cases in which hydrocephalus simulates brain tumour. The X-ray pictures, to be of most service in such cases, should be made stereoscopically, as Dr Leonard's plates were made in this way, and thereby the recognition of the condition was made easier. His photographs will be published in the *Archives of the Roentgen Ray.*

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[1] Gordon, *Journal of Nervous and Mental Disease*, Sept. 1910, p. 558.

[2] Since the above was written Dr Leonard has made an X-ray photograph of the calvarium from my patient, and has obtained a picture like that described in this article.

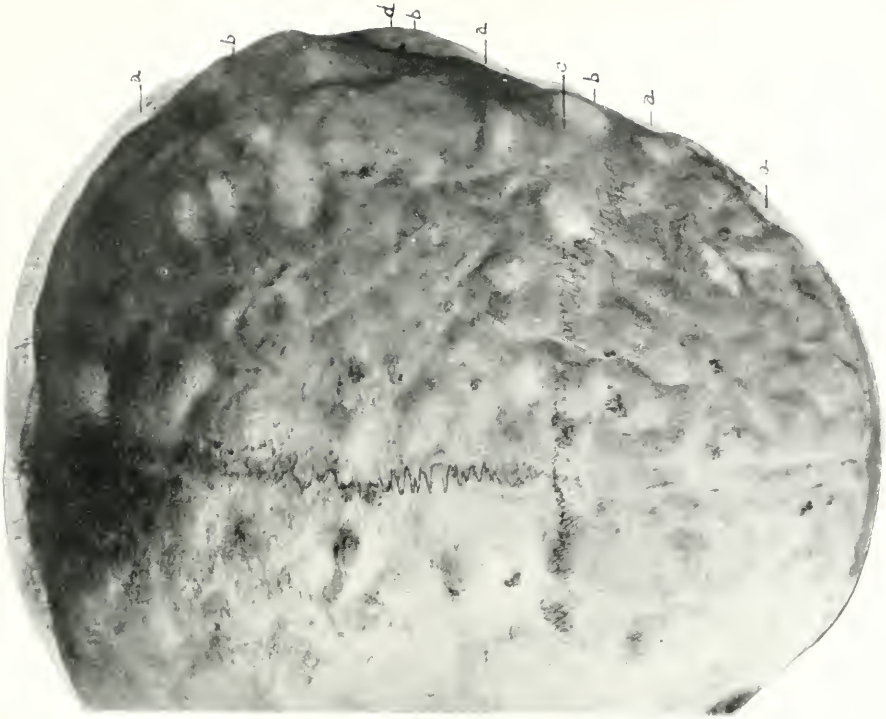

FIG. 1.

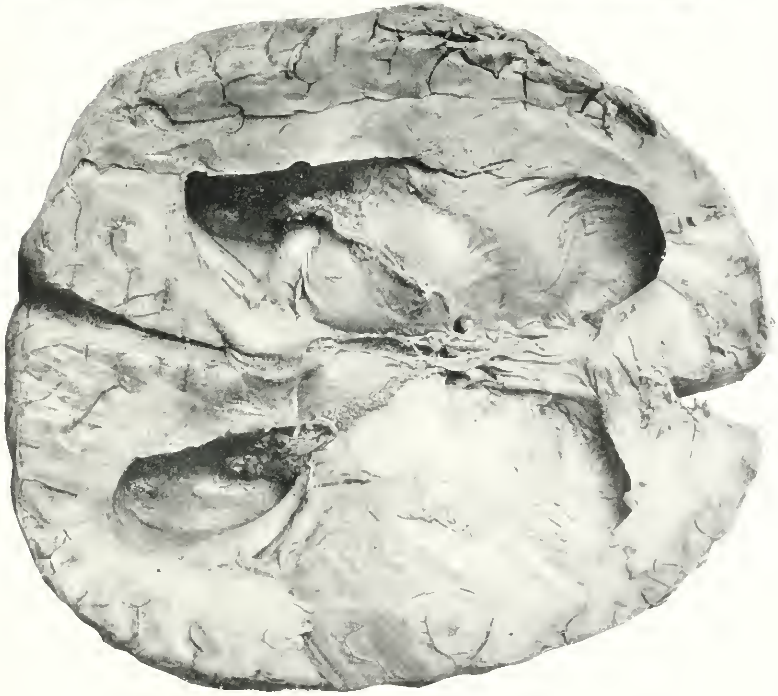

FIG. 2.

To illustrate Dr Spiller's Paper

Description of Figures.

Fig. 1.—The photograph represents the atrophied areas somewhat inadequately. The white margin (*a a a a*) represents the thickness of the bone where the saw has cut. At places (*b b b*) where the saw has cut through atrophied areas, the bone is not thicker than a piece of paper. The bone in some of the depressions, as at *c*, is so thin that the finger placed on one side of the calvarium may be seen from the other side as a deep shadow.

The distance from the angle *d* to the opposite angle of the calvarium, made by the saw, is 15 cm. The widest transverse diameter of the calvarium is 16 cm. The longest antero-posterior diameter is 18 cm. The cut of the saw posteriorly passes through the upper part of the lambdoid suture ; anteriorly it is 9 cm. from the highest part of the coronal suture.

Fig. 2.—Photograph showing the pronounced hydrocephalus which caused the atrophy of the inner table of the skull in areas corresponding to the cerebral convolutions.

Postoperative Psychoses.

By JOHN H. W. RHEIN, M.D.,
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(Read before the Philadelphia Psychiatric Society, May 19, 1911.)

Reprinted from Pennsylvania Medical Journal, September, 1911.

While psychoses after operations are not very common, they occur with sufficient frequency in the experience of surgeons to warrant a more careful study on the part of the psychiatrist than they appear to have been accorded hitherto. Textbooks on psychiatry on the whole have neglected this subject in large measure, most of the literature or a great part of it having emanated from the surgeon and therefore studies of postoperative psychoses, along the lines of modern psychiatry, have been lacking to a great extent.

The first description of this condition dates back to the sixteenth century when Paré[1] called attention to the occurrence of mental symptoms after operations. In 1819 Dupuytren[2] described a delirium nervosum which occurred in the first three days after an operation, and in 1842 psychoses after eye operations were reported by Herzog.[3] A very extensive résumé of the history of postoperative psychoses may be found in the excellent publications of Picque,[4] Schultze[5] and Engelhardt,[6] a review of which shows that this subject has been extensively treated in the literature.

While the cases which are reported in this paper are in no sense unique, they offer an opportunity of discussing some of the features of the subject, which may prove interesting if not instructive.

In studying the effect of an operation upon the mental condition, numerous etiological factors are brought into consideration. First of all, the hereditary predisposition of the individual operated upon, his habits as to alcohol and drugs, his physical, mental and moral qualities, the moral attitude assumed upon entering upon the operation, effect of anesthetics, dressings, and complications of the operation, such as infections, all deserve serious considerations. All of these elements probably have a greater bearing on the occurrence of abnormal mental states after operation than the character of the operation itself. It is generally agreed, I believe at present, that the character of the operation in itself has little or no influence in inducing a postoperative psychosis.

It has been contended in the past that serious operations upon the pelvic and genital organs and operations upon cataracts were especially apt to be followed by mental disorders, but recent authorities contradict this opinion (Joffroy,[7] Selberg,[8] Lemesle,[9] Rohé[10] and Lapinsky[11]).

At present the general view, and I believe the correct one, held as to the cause of insanity after cataract operations, is that it is due to cerebral arteriosclerosis, most if not all of such cases occurring in old people (Lapinsky, Schnabel[12]). The effect of the darkness of the room has little weight it is claimed, as the delirium has been observed to occur frequently after the removal of the bandages, while Krafft-Ebing[13] believed that the effect of the dark room was by reason of fright.

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[1] Paré: In Schultze.

[2] Dupuytren: In Engelhardt.

[3] Herzog: In Engelhardt.

[4] Picqué: *Bull. et mém. Soc. de chir. de Par.*, 1898, No. 24, p. 171.

[5] Schultze: *Deutsch. Zeitschr. f Chir.*, 1910, p 584.

[6] Engelhardt: *Deutsch. Zeitschr. f. Chir.*, 1901, Vol. LVIII., p. 46.

[7] Joffroy: *LaPresse Médicale*, 1898, No. 24, p. 141.

[8] Selberg: Bruns' *Beiträge zur klin. Chir.*, 1904, Vol XLIV., p. 173.

[9] Lemesle: Psychoses post-opératoires, 1900, These, Paris.

[10] Rohé: *Am. Jour. Obst.*, 1899, Vol. XXXIX., p. 324.

[11] Lapinsky: *Allgemeine Zeit. f. Psychiat.*, 1906, Vol. LXIII., p. 665.

[12] Schnabel: In Engelhardt.

Those cases developing immediately or very shortly after operation, associated with febrile reaction or disturbance of kidney or liver functions or toxic or infectious states, are interesting and perhaps the most frequent and characteristic forms of psychoses, and are readily explained on the basis of toxic or septic origin. These patients usually exhibit symptoms of melancholia, toxic delirium or maniacal excitement and are directly related to the conditions which arise in the nature of complications of an operation; for example:—

A woman of thirty-two years was operated on at the Howard Hospital for stone in the kidney. There was a marked febrile reaction for nine days after the operation, due to a bronchopneumonia. On the fifth day after the operation the patient became very depressed and hypochondriacal. She thought she was going to die, that there was no hope for her recovery; she did not wish to live, presented the appearance of abject depression, was unreasonable, refused to see her husband for whom she had apparently lost her affection, and moaned constantly. At the end of four weeks the mental condition cleared up entirely. There was associated with this condition disturbance of the kidney functions, the urine containing casts and albumin.

A second case in point showed symptoms of acute delirium and maniacal excitement. The patient, a woman of twenty-eight, was operated on at the Howard Hospital, a double Edebohls, ventral suspension of the uterus and appendectomy having been performed. The temperature remained normal until the seventh day and then followed an irregular course, never rising above 101, and returned to normal on the seventeenth day. Two days before the rise of temperature she became slightly delirious, talked incessantly and incoherently. About nine days later she became very noisy, moaning and crying and was delusional. This condition alternated with periods in which she would not answer questions and remained quiet with eyes wide open and fixed. At the end of twenty days the mind began to clear and she rapidly recovered. There was no evidence of septic infection in this case and the only perversion of function present was that of the kidneys, the urine showing casts and albumin, which persisted for a time after the mental condition cleared up.

Those cases which occur several weeks or months after an operation should not be included among the postoperative psychoses, for example:—

A patient was admitted to the insane department of the Philadelphia Hospital, February 20, 1902, with a history that the first symptoms began a month previously, January 18, 1902. Two months prior to this she had had some gynecological operation performed but had exhibited no mental symptoms until two months later. She developed a chronic mental state in which she was disoriented, had delusions of persecution, refused to eat or talk, was depressed and delusional and required restraint. She died at the end of two years uncured. In this case I do not believe that the operation had any direct bearing upon the development of the mental state.

Many cases similar to this are described as postoperative psychoses and I believe incorrectly. Moreover, a careful study of these cases will show in a certain percentage that mental symptoms were present before the operation, after which the symptoms may become more apparent or perhaps aggravated; for example:—

A man of forty-eight, was admitted to the insane department of the Philadelphia Hospital, exhibiting delusions of grandeur, irritability, excitability to the point of violence, loss of memory and insomnia. He was admitted to the hospital three weeks after an operation for depressed fracture of the skull, the result of a fall three months previously. A careful study of the history revealed the fact that these symptoms had already begun to make their appearance shortly before the accident, though much aggravated after the operation, and were really in no way directly related to the operation itself.

Cases occurring within a few days after operation, which do not show hereditary predisposition; complications of a septic or toxic nature; disturbance of kidneys or liver functions; or cases in which history of alcoholism and hysteria can be eliminated, must have some relation to the operation itself. Whether these cases are a result of the traumatism of the operation or are coincidental, or what not, is still open to discussion.

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[13] Krafft-Ebing: Textbook on Insanity (Chaddock). 1904, p. 166.

The following cases are worthy of brief report as showing an absence of etiological factors outside of the operation itself.

R. S., aged twenty-four, was admitted to the insane department of the Philadelphia Hospital, May 16, 1904. The family and previous histories were negative. His first symptoms began a week after an uncomplicated operation for appendicitis. The physical examination was negative upon admission. He believed he was dying; he was noisy, cried and talked to himself. He believed that he was to be operated on again, that someone was going to hurt him, that people were saying strange things about him and that they followed him around by day and night. At night he heard the voices of people whom he had seen during the day. The voices said to him, "Budda lies without. Is he out? Is he in?" He was coherent, quiet and obedient otherwise. At the end of four months' time he returned to his home entirely recovered.

E. C., a woman of fifty-two, was admitted to the Philadelphia Hospital, September 11, 1908, with negative family and previous histories. Two weeks after vaginal hysterectomy, convalescence from which presented no complications, she became delusional and had hallucinations of sight and hearing, refused medicine and food, and believed that she had committed the unpardonable sin. Upon admission her physical examination was practically negative. She was very sad and depressed and talked very little. She imagined that everyone was a detective and that her brother had stolen a lot of money. She was mentally dull, talked to herself, showed considerable mental confusion for remote and recent events and had suicidal ideas. At the end of a year she recovered.

A. G., a man of forty-three, presented a negative family history. His symptoms dated from an operation upon a rectal abscess in the autumn of 1904. He became depressed and sleepless and was unable to work. He stated that his head was confused. He was sad and moped all day with his head in his hands. He was melancholy and believed that everyone was abusing him and that he would never recover. He attempted suicide on one occasion. Physically there was nothing abnormal except that urinary analysis showed the presence of casts. He completely recovered at the end of three years.

K. F., a woman of forty-five, after an operation for hemorrhoids and fistula, became greatly depressed and wandered the streets, weeping constantly and believing that she would never recover. This she blamed upon the operation. Upon admission to the Philadelphia Hospital, January 12, 1899, she presented a sad depressed appearance, was anemic and emaciated, complained of throbbing headaches vertigo and constipation. She had auditory hallucinations and delusions of persecution, heard voices calling her by her maiden name, talked a great deal about the operation and showed anxiety about the condition of her bowels. She believed that she was dying and begged not to be killed. She was discharged cured at the end of three months.

These cases are difficult ones to explain etiologically. Some element connected with the operation must have some direct bearing upon the immediate appearance of the symptoms. If it were possible to explain the causes of melancholia in general, it might be easier to understand why an operation should give rise to the development of postoperative psychic states. Certain it is, that an operation simple or complicated, even when the shock is avoided by a properly administered anesthetic, causes a profound impression upon the nervous system of a detrimental character. Why it should cause in some neurasthenia, or hypochondriacal tendencies, and in others more profound injury to the psychic state to the point of mental unbalance, is yet to be explained, but I am convinced that there is something in the mere physical assault of the surgeon's knife that may influence the development of a psychosis. In this connection should be mentioned the statement of Schultz, that there is no operation which gives no effect upon the psychic sphere. While Truelle[14] did not believe that these cases should be called instances of postoperative psychoses and only gave the operation the same importance that would be attached to reverse of fortune, a moral shock, or the like, Engelhardt was of the opinion that there are cases of postoperative psychoses which can not be etiologically explained, and Guyot[15] asserted that, while their existence has been contested because they do not form a group in themselves, this does not exclude their existence. Furthermore, Urbach[16] did not think that acute psychoses after operation on the gall bladder were accidental.

When the cases show complications, such as a history of alcoholism or syphilis or other infections, the etiology becomes readily apparent; for example:—

James K., an adult male, was a heavy drinker. Two and a half months previous to his admission to the insane department of the Philadelphia Hospital, he was operated on for hernia, making a prompt and good recovery. Immediately after his discharge from the hospital he became despondent and depressed, seclusive and secretive. He was restless and sleepless, complaining of pain in the head and stomach. He would grind his teeth and cry out without apparent cause, and wandered about the streets aimlessly. He would frequently cry out, "I have sinned a great deal," and was distinctly emotional and tearful. He was somewhat confused and ideation was delayed. There was some hallucination of sight, such as seeing his brother holding before him the image of Christ in his hand. This patient was discharged cured in six weeks' time. The diagnosis in this case was alcoholic confusional insanity, the operation having only a purely incidental significance.

Another instance illustrating this point was as follows:—

A young woman had been admitted to the surgical ward of the Philadelphia Hospital with a diagnosis of secondary syphilis. She was transferred to the gynecological ward and a hysterosalpingo-oöphorectomy performed on November 4, 1910. In five days' time the patient began to be noisy, excited and restless, requiring restraint. She used vile language and talked disconnectedly. She saw the devil and black cats and heard voices. She was elated and answered questions in a silly manner, was profane and obscene, was filthy about her person and talked wildly, rapidly and disconnectedly. The physical examination was practically negative. While there was no hereditary predisposition, she had always been a wayward and disobedient child, inclined to be on the streets at night and to remain away from home for several days at a time. Her habits as to alcohol and tobacco could not be determined, but it was evident that her mode of life was irregular and immoral. The effect of an operation upon such a case can be only incidental.

As in the foregoing case, there was an absence of heredity in all these cases and I think one goes too far to say, as has been claimed by Truelle, that hereditary predisposition is present in most of the observations.

The types of psychoses which are more frequently met with after operations, are melancholia, mania or hallucinatory or confusional forms of insanity. In my own experience depressive forms of psychoses were the most common. According to some, confusional insanity was the type in the majority of cases. Selberg describes an acute hallucinatory insanity in which the patient is disoriented, depressed and suicidal, exhibits great motor unrest and in which there are numerous hallucinations present. Marlier[17] found that mania and melancholia were the common forms in his experience. Some authorities contend that there is no special type of insanity after operation (Rohé, Joffroy, Truelle, Reyneau[18] and Fenayrow[19]), although Werth[20] believes that these cases are usually of the type of melancholia, while in Karlsberg's[21] opinion they are chiefly of hallucinatory form.

I wish to express my thanks to Dr. Allan Jackson for his kindness in permitting me to make use of the cases from the insane department of the Philadelphia Hospital.

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[14] Truelle: Psychoses dites post-opératoires. 1899. These, Paris.

[15] Guyot: Psychoses post-opératoires, 1899, These, Paris.

[16] Urbach: *Wien. klin. Wochenschr.*, 1907, No. 47, p. 1465.

[17] Marlier: La Folie post-operatoire, 1897, These, Paris.

[18] Reyneau: In Fenayrow.

[19] Fenayrow: *Arch. Neurol.*, 1889, p 257.

[20] Werth: *Centralbl. f. Gynak.*, 1888, No. 121, p. 387.

[21] Karlsberg: *Ibid.*, p. 692.

Extracted from the American Journal of the Medical Sciences, January, 1911

PELLAGRA, WITH A REPORT OF TWO CASES WITH NECROPSY.[1]

By Paul V. Anderson, M.D.,
OF MORGANTON, N. C.,
AND
William G. Spiller, M.D.,
OF PHILADELPHIA.

Clinical Report by Dr. Anderson. Case I —Mrs. D. E., white, aged twenty-two years. The family history is negative except for the fact that one brother died with pulmonary tuberculosis. The patient had the diseases common to childhood. Catamenia occurred at sixteen, and was regular, but has been absent since March, 1909. She has always been in good health until four years ago, when she began to have gastric disturbance. This began in the spring, but became less severe during the fall. The following year, in March, her gastric symptoms returned and she had a persistent diarrhœa, with several watery stools a day. Later, the dorsal aspect of her hands and wrists became reddened and the palms roughened. There was a reddened patch on the neck, at the root of the hair, and on the nose and at the corners of the mouth. The patient states concerning her hands: "They were red, itched, and were sore, and looked just like they had been dipped in hot water and scalded." The elbows were also red and roughened. The mouth and tongue were sore. Salivation was profuse. Hunger and thirst were excessive, and there was considerable loss of weight. She was able to do her housework, however, and was not confined to bed. After a short time the skin of the hands and of the patches on the nose and neck peeled off. The diarrhœa gradually grew better, and in the fall the patient gained flesh and seemed to be in fairly good health. In March of the following year the eruption again appeared as before, accompanied by the symptoms previously enumerated, but they were more severe, especially the diarrhœa, and the patient became so ill that she was confined to her bed. In August, 1909, she was taken to a general hospital, where she remained for a month. When she left the hospital she was better; the skin of her hands had again peeled, but the hands were roughened. She was unable to take up her housework, however, on account of weakness and nervousness. She also was not able to walk well, and at times felt dizzy and blind.

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[1] Read at a meeting of the College of Physicians of Philadelphia, December 7, 1910.

The patient became disturbed mentally in January, 1910, and was admitted to the Western North Carolina Hospital for the Insane February 27, 1910. The duration of the mental trouble before admission was one month. Prior to admission she was sleepless, restless, apprehensive of bodily harm when left alone, silly in speech and behavior, and had hallucinations of sight. At one time she threatened suicide. The physician who committed the patient made a diagnosis of "neurasthenia bordering on melancholia." (This diagnosis has frequently been made in cases which later proved to be pellagra.)

Since the patient has been in the hospital she has been emotionally unstable, weeping without the slightest cause, and occasionally smiling in a wan way. As a rule, however, she has an anxious, apprehensive expression on her face. She says that her blood and nerves are wrong; that her uterus and stomach are terribly diseased. She complains of excessive pains in her ankles and knees. She says, also, that her back is weak and causes her pain. There is marked salivation—in fact, so great is this that her pillow is frequently wet by it. Her appetite is ravenous and her thirst is excessive. Her mouth and tongue are sore and she complains of a burning sensation in the œsophagus and stomach. She is constantly complaining of these symptoms and begs the doctor to give her medicine for them, as long as he is in sight. She also begs him pitifully not to let her die. In answering questions she is more or less confused, at times, and her memory is defective. She speaks in a monotonous voice, never varying the tone. She walks with difficulty, but for a few days after admission she was able to walk from her ward to the porch without assistance.

Physical Examination, March 19, 1910: The patient (Fig. 1) is well developed, but very much emaciated. She has an anxious, agitated expression on her face and cries and moans almost constantly, bewailing her fate. At times she has a mask-like expression of the face. There are no asymmetries of face. The ears are well formed; lobules attached. The pupils are regular; contracted; react sluggishly to light and to accommodation. There is no nystagmus and no strabismus. The conjunctivæ are sensitive to touch. The tongue is protruded straight, but marked coarse tremors are present. The edges of the tongue are reddened and the central part is covered with a brownish coating. Salivation is present to a marked degree. There is a reddish-brown discoloration extending from the base of the neck upward to the hair-line, and there is also a reddish purplish discoloration on the dorsal aspect of the hands, extending an inch or two above the wrist (Figs. 2 and 3). This is symmetrical on

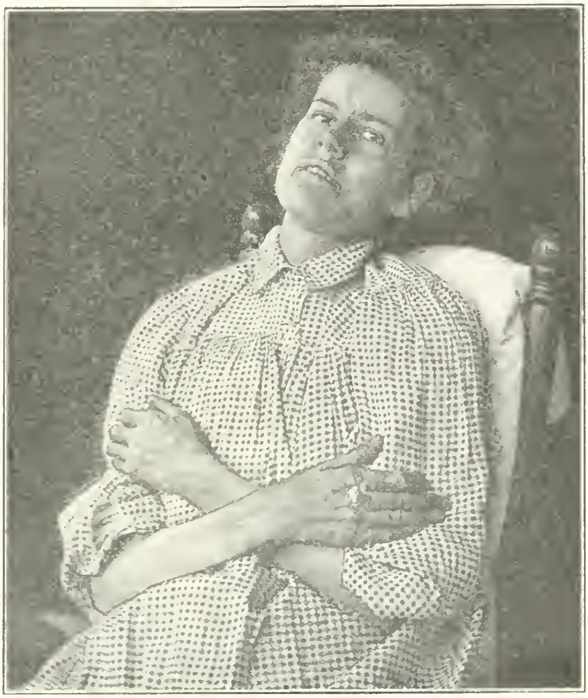

FIG. 1.—Photograph of the patient (Case I), showing the retraction of the head.

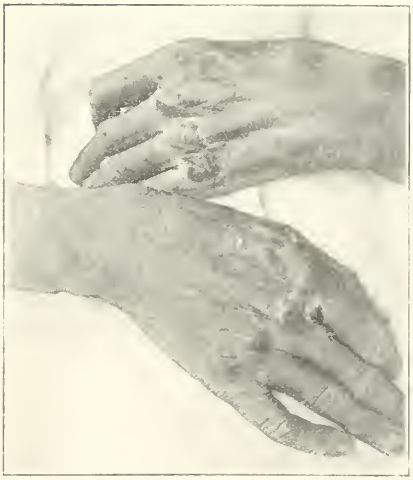

FIG. 2.—Photograph of the hands, showing the pellagrous eczema on the dorsal surfaces.

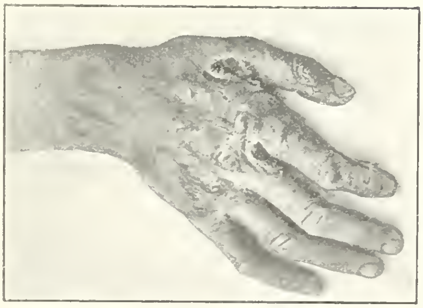

FIG. 3.—Photograph of the right hand alone, showing the eczema more distinctly.

each side. Nothing abnormal is noticed about the palms. The normal lines of the skin on the dorsal aspect of the hands seem to be lacking. The chest is symmetrical, more or less flattened, and the subclavicular fossæ show a more or less marked depression on each side. No abnormality can be determined by percussion, but auscultation reveals harsh breath sounds at the left apex. The heart is apparently normal in size and no murmurs can be heard. The abdomen is slightly distended. No masses can be felt. The abdominal organs are apparently normal. The liver is contracted. It extends from the fourth interspace to the seventh rib in the midaxillary line. The abdominal reflexes are absent. The plantar reflexes are greatly exaggerated. The Babinski reflex and ankle clonus are not obtained. The patellar reflex is greatly exaggerated. The patient complains of excessive pain when she is tested for ankle clonus. There is a marked, coarse tremor of the head, and movements of the hands and arms are more or less athetoid or ataxic. There is marked incoördination in the upper extremities, the patient being unable to place her hand on her nose with the eyes shut. The station is poor; the patient shows a tendency to sway and would doubtless fall if she were not caught when the eyes are closed. The gait is more or less spastic and uncertain. She cannot walk without assistance, and walks with the feet spread wide apart. Apparently she has difficulty in recognizing the difference in the heat and cold tests on the feet and legs, but she could tell the difference when the tests were applied to her abdomen. The test for touch and pain could not be carried out on account of the patient's failure to coöperate. There are many coarse tremors of the hands when they are extended.

Smell: Turpentine; castor oil; ammonia, not recognized; camphor, correct.

Taste: Sour, correct; salt, correct; bitter, sour; sweet, not recognized.

March 30, 1910. Numerous bullæ have developed on the dorsal aspect of patient's hands, and in places these have broken, leaving sores. There is a marked reddening all over the dorsal aspect of the hands. There is also marked scaliness with excessive redness of the forehead, on the nose, and on the back of the neck. The elbows are very much roughened. The patient is quite emotionally unstable. She is fearful that she will die, and cries a great deal of the time. She almost invariably has an anxious, set expression on her face, and although, at times, she laughs in a silly way, her expression is more or less fixed all the time. There is marked redness of the mucous membrane of the mouth; the tongue is reddened and fissured, and there is an ulcer at each corner of the mouth. There is also marked salivation. She walks with difficulty—in fact, she cannot walk alone at all. The patellar reflex is greatly exaggerated. She eats ravenously and is excessively thirsty. She also, at times, has slight opisthotonos, and complains of excessive pains in her legs. She also states that her hands burn terribly. Her bowels are constipated. She sleeps scarcely at all.

April 15, 1910. The patient is growing much weaker. She has a persistent diarrhœa, with several watery, foul-smelling stools a day. She is excessively nervous and her muscles twitch a great deal. This condition exists even in her sleep. She is greatly depressed and is apprehensive that something terrible is going to happen to her. She cries a great deal and begs constantly for food and water. The deep reflexes are still greatly exaggerated. The opisthotonos is increasing in severity. She cannot walk at all and has to be carried to her chair on the porch.

May 1, 1910. The patient is confined to bed. There are marked clonic convulsive movements of her legs and arms, and twitchings of her whole body; in fact, she is scarcely still a minute. The slightest touch produces a marked convulsive movement. She is still excessively thirsty and eats ravenously. She has a persistent diarrhœa, with numerous watery, foul-smelling stools each day. The dorsal aspect of her hands presents a reddish, purplish appearance. The epidermis is coming away in scales. There is also a scaliness about the corners of the mouth, on her nose and forehead, on the back of her neck, and on her elbows.

May 8, 1910. The patient is much weaker. Speech is thick and she expresses herself with difficulty. At times she is delirious. There is a very marked opisthotonos and there are clonic convulsive movements of the arms and legs. At times there are tonic spasms, and marked muscular twitchings are present most of the time. The mouth is excessively reddened, with saliva drooling from it, and the tongue is beefy in appearance. The deep reflexes are still greatly exaggerated, but are not so much exaggerated as they were for a time.

May 9, 1910. The patient died today.

March 1, 1910. Urinalysis: Straw-colored, flocculent precipitate; slightly acid; 1010; trace of albumin; no sugar; no casts; many urates; a few epithelial cells.

March 3, 1910. Examination of sputum for tubercle bacilli, positive.

April 23, 1910. Urinalysis: Light amber; clear; acid; 1013; no albumin; no sugar; no casts; many epithelial cells.

April 23, 1910. Feces examined—no hookworm ova found.

Case II.—E. A. H. (under the care of Dr. Jas. K. Hall. Notes by Dr. Hall).

This man was admitted to the Western North Carolina Insane Asylum in 1888, at the age of forty, from a county in the extreme western portion of the State. He had been married for ten years and had a wife and two children. He was a gunsmith and cabinetmaker, but he had no education and was without property. For ten years or more before admission he had been wandering about over the neighborhood, had done little work, and for perhaps more than ten years he had been looked upon as mentally abnormal. At times he was mute for days; sometimes he was extremely irritable, had visual hallucinations, and he occasionally threatened violence to members of his family and his neighbors. He attempted suicide, and pending admission to the hospital he was kept in the county jail for several weeks.

When admitted to the hospital the patient was a quiet dement, in rather frail physical health. Two years after admission he had a severe attack of influenza, but he made a good recovery, and six years later he had measles. When able, he worked on the farm, but there was a loud, systolic murmur, and at times the feet became so swollen that confinement to bed was necessary for a short time. In the latter part of 1903 he was transferred from the main building of the hospital to the colony, where the patients all engage in outdoor work, and this man found employment in breaking stones with a hammer, for macadamizing the roads. The note was made that his physical health during the winter of 1903-04 was first-rate, but a line in his history, dated April 1, 1904, states that he has an eczema-like eruption on his hands, and for that reason he is left indoors. On June 16, 1904, it was noted that he had been in bed for several days because of disturbances of cardiac compensation; that he had been unusually depressed and ate little, but the condition of the hands had improved. In the early part of October he was said to be very weak, emaciated, and there were bedsores. The physical condition had improved by the spring of 1905, and he was again able to be out of bed. For the following two years he remained in the hospital building, doing a little work in the ward. and it is presumed that his physical health was fairly good. inasmuch as no mention is made of illness in the history On May 15, 1907, the following note was made: "This patient was transferred from the hospital building to the colony about April 1; he busies himself in helping to keep one of the buildings in order, but he is profoundly demented, and the scope of his intellectual activity is extremely circumscribed. It is impossible to induce him to raise his voice and he speaks always in a whisper. His general health is not robust, but apparently fairly good." The old man remained at this colony until September 24, 1909, when he was again placed in a hospital ward because of failing physical health. At that time the backs of the hands looked as if they had been badly sunburned. The bowels were loose, the appetite was failing, and he was losing in weight. The gait was uncertain and tottering; the movements of the upper extremities were athetoid; the protruded tongue was tremulous, and the deep reflexes were exaggerated. Ankle clonus and Babinski's sign were absent. No doubt was felt that the condition was due to pellagra.

The decline was steady and progressive. The diet was restricted principally to milk, and although sufficient nourishment was taken, emaciation increased. Dementia was so far advanced that no intelligent statements could be elicited from the patient. Diarrhœa was troublesome, and all control of the movements was soon lost. Tonics were given and efforts were made to control the diarrhœa, but without avail. As the emaciation increased, bedsores developed. The bed was soiled every few minutes. The bowel movements were a dark, grayish liquid, and peculiarly offensive. The lower extremities became drawn, and efforts to extend them seemed to cause great pain. There were almost constant, apparently purposeless, movements of the upper extremities, extremely awkward and incoördinate. He was extremely restless and his behavior induced the belief that he was annoyed by visual hallucinations. As the disease progressed, the muscular disturbance became more marked. In addition to the gross movements of the upper extremities, there were spasmodic movements of the fingers. The eyelids frequently moved rapidly. At times there were clonic, convulsive-like movements of the whole body, of momentary duration. Not infrequently an unusually loud inspiratory grunt could be heard, due probably to spasmodic contraction of the diaphragm and other inspiratory muscles. Firm pressure on the skin seemed not to produce pain, but the gentlest tap on any part of the surface caused generalized, clonic muscular spasm. Pupillary abnormality was not noted. The temperature was never much above normal and in the last days it was slightly subnormal. The pulse became quite small and rapid. Urinalysis showed a trace of albumin and a few hyaline casts. Repeated differential blood counts invariably showed an increase in the number of large lymphocytes. One count gave the following result: Polymorphonuclear neutrophiles, 52.75 per cent.; eosinophiles, 2.5 per cent.; large lymphocytes, 22.75 per cent.; small lymphocytes, 22 per cent.

Death occurred January 3, 1910.

The necropsy showed nothing unusual. Emaciation was extreme; the muscles were quite red; the lungs and pleuræ were normal; the heart was somewhat enlarged, and the mitral leaflets were gnarled. The spleen was small, dark, and tough; the liver was about normal in size and consistence, and the intestinal wall was thin and pale. The kidneys were small; the capsule was adherent; the organs were tough and the cortex shrunken.

Remarks. It will be observed that this man was an inmate of the institution for twenty-two years before his death. During this time he had lived a sheltered, protected life, in a climate noted for its healthfulness. He was probably the first patient in the hospital to develop pellagra. It is scarcely to be doubted that the soreness of the backs of his hands noted in 1904 was the dermatitis of pellagra. For the following five years there were apparently no symptoms of the disease. The final attack developed late in the summer. For more than two years before his last illness he lived in a colony with seventy-five other patients, almost a mile from the hospital, and during this time he was not near any pellagrous patients. His diet and his hygienic conditions were exactly those of a thousand other patients. Contagion was not possible. If the food and surrounding conditions caused the disease it seems strange that he alone should have been affected.

THE PATHOLOGY OF PELLAGRA BY DR. SPILLER. The number of clinical cases of pellagra reported in this country has become quite large since the disease was first recognized here a few years ago. So far as I know, no report of a necropsy in any of these cases has been published, and it is important to determine whether the pathology of the American cases conforms to that of the European or not. It is presumable, however, as the symptoms of the disease are essentially the same in our country and in foreign lands, that the pathology will not vary greatly. Pathological reports are found chiefly in the Italian language, and the only one in English known to me is by Batten, who studied the spinal cord in three cases described by Sandwich.[2] Batten found degeneration of the posterior columns in two cases, but the third case showed practically no change. He believed the sclerosis of the posterior columns to be of root origin. The process was a chronic one, as shown by the fact that the posterior columns were extensively sclerosed and no degeneration was revealed by the Marchi stain. This report by Batten is especially interesting in that it seems to show that the degeneration in the spinal cord was not rapidly progressive, otherwise changes would have been detected by the Marchi stain.

Tanzi,[3] in the recent English translation of his text-book, says that Tonnini, in 1883–1884, was the first to associate the spinal phenomena of pellagra with primary spinal lesions (cellular pigmentation) of the anterior and posterior horns of the spinal cord. In cases of long standing there may be general or local œdema of the brain, hyperemia, and thickening of the meninges, chronic internal hydrocephalus, and sometimes softening of the cerebral substance and hemorrhages. Accumulations of pigment are also to be observed in the cells of the spinal cord and in those of the sympathetic ganglia. The spinal cord at an early stage exhibits traces of systemic degeneration of combined type, that is, lesions of the posterior columns and of the crossed pyramidal tracts, as has been shown by Tuczek and Belmondo. There is also very often small-cell infiltration of the spinal meninges. In the acute cases the nerve cells of the cerebral cortex are swollen; in some instances the nucleus is displaced to the periphery, the processes are atrophic, and there is central chromatolysis.

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[2] Jour. of Path. and Bact., 1901, vii, 460.

[3] A Text-book of Mental Diseases, translated by W. Ford Robertson and T. C. Mackenzie, Rebman Co., New York.

Babés and Sion,[4] in their monograph on pellagra, state that in most cases of this disease nothing peculiar was found in the spinal roots, but in other cases notable degenerative changes were found in the posterior roots. In the spinal cord were pronounced pigmentation of the nerve cells of the anterior and posterior horns, and often combined sclerosis.

Babés and Sion, in their own cases, found the posterior roots often degenerated, and the degeneration of the posterior columns was secondary to this, but differed from that of tabes in the less involvement of Lissauer's zone and of the anterior root zone, unless the pellagrous changes in the posterior columns were far advanced, when these zones also were degenerated. The cells of Clarke's column were always affected. The cells of the anterior horns were degenerated, as were also the cells of the cerebral cortex. They regard the changes as produced by some poison.

Lukács and Fabinyi,[5] in a recent paper, state that system degeneration of the spinal cord is rare in pellagra. The cerebral meninges in their cases were intact, but in one case the spinal pia was a little thickened in places and infiltrated with cells. They found no unusual arteriosclerosis, although Marianis records arteriosclerosis as constant in pellagra. The medullated fibers of the brain, cerebellum, and medulla oblongata were normal (Marchi's method, however, was not used). In two cases the spinal tracts were intact, in a third the columns of Goll were degenerated from the pyramidal decussation to the upper thoracic cord. Many cells of the posterior horns were degenerated from the cervical swelling downward, but less intensely in lower levels, and this cellular degeneration is supposed by the writers to have been the cause of the degeneration of the columns of Goll. Degeneration of these columns, they state, is very common in pellagra, and differs from that of tabes in that it is seen only in the cervical region, and in that Lissauer's zone is intact. The cells of Clarke's column were greatly degenerated. Nuclei of sensory nerves in the medulla oblongata and pons were scarcely altered, while motor nuclei here were intensely affected. The cells of the anterior horns were greatly degenerated, especially in the cervical region, and showed granular chromophilic elements at the periphery, and a homogeneous centre, with absence of nucleus. The cells of the brain cortex were likewise affected, and especially those in the central convolutions. The lesions, they state, correspond to those of ergotin, mercury, or aconite poisoning, and pellagra is a disease of toxic nature.

Parhon and Papinian[6] seem to be the only investigators who have studied the neurofibrils in pellagra. They found the alterations less intense in the small cortical cells of the brain, and most intense in the Betz cells. The neurofibrils stained less deeply, were thinner, even fragmented. In some small pyramid cells they were not altered. In the large cells the neurofibrils were almost completely absent; in some they were preserved at the periphery and in the cell processes. Alterations were more intense in the cervical region than in the lumbo-sacral region, and were of the same kind as in the Betz cells.

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[4] Nothnagel's System.

[5] Allg. Zeitsch. f. Psych., 1908, lxv, 657.

[6] Comptes-rend. de la Soc. de biologie, 1905, lviii, 360.

Pathological Report of Case I (Case of Dr. Anderson). The nerve cells of the anterior horns of the lumbar region are intensely degenerated. The cell body is swollen, the nucleus is displaced to the periphery, the dendritic processes of many cells have disappeared, and the cell body shows intense chromatolysis, sometimes implicating the whole cell, sometimes leaving a zone of chromophilic elements at the periphery of the cell body. The cells of the anterior horns stained by the Bielschowsky silver stain show an absence or a fragmentation of the neurofibrils. There is no round-cell infiltration of the pia. The anterior and posterior roots appear to be normal. Marchi's stain reveals diffuse degeneration in the posterior and antero-lateral columns, but less in the posterior columns near the gray matter. Numerous holes, from which nerve fibers have disappeared, are found in the antero-lateral columns. The

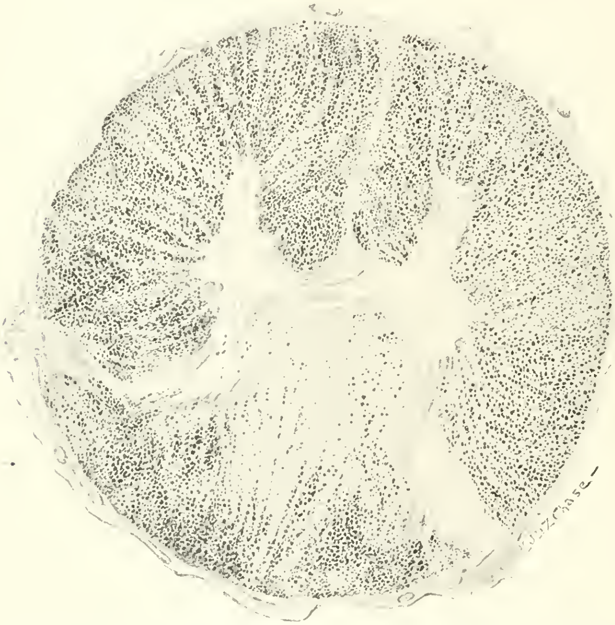

FIG. 4.—Drawing of a section of the thoracic cord from Case I, showing the diffuse degeneration of the posterior and lateral columns by the Marchi stain.

pia is not thickened and the bloodvessels are not sclerotic. The thoracic cord is much distorted, probably as a result of injury at the necropsy. Here and there a swollen axis cylinder may be found in the antero-lateral columns. The cells of the anterior horns present the same condition as do those in the lumbar region. The cells of the columns of Clarke show similar changes. The degeneration of the posterior and antero-lateral columns by the Marchi stain is pronounced (Fig. 4). Only the lowest part of the cervical cord was obtained. The anterior horn cells are in the same condition as are those in the lumbar region. The anterior and posterior roots of the eighth cervical and first thoracic segments, cut separately from the cord, appear to be normal. The capsules of the cells of the lumbar posterior ganglia show much proliferation of their lining cells, and the nerve cells are in part degenerated. Cells of the hypoglossus nucleus present a condition similar to that of the cells in the anterior horns, but of less intensity. The cells of Betz and the nerve cells of the deeper cortical layers in the paracentral lobules show changes similar to those of the anterior horn cells, that is, swelling, chromatolysis, and displacement of the nucleus (Fig. 5).

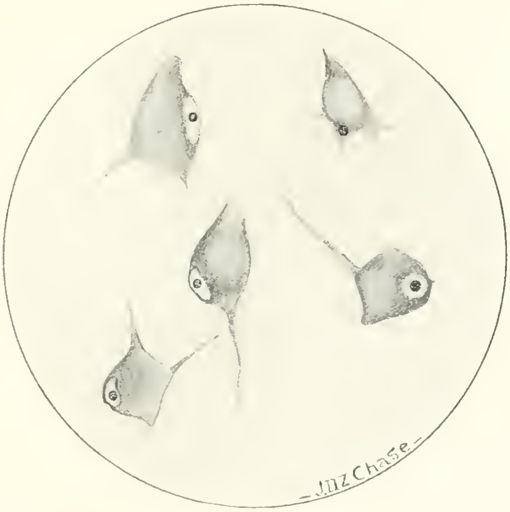

Fig. 5.—Drawing of nerve cells from the paracentral lobule and the anterior horns of the lumbar region. Cell of Betz from the paracentral lobule. One of the larger pyramidal cells of the deeper layers of the paracentral lobule. Cells of the anterior horn of the lumbar region. All these cells are much degenerated.

Pathological Report of Case II (Case of Dr. Hall). Only a small piece of the spinal cord was obtained, and this was from the thoracic region. The pia is distinctly thickened. Diffuse but slight proliferation is found in the lateral and posterior columns; the cells of the columns of Clarke are much swollen; the nucleus is eccentric, and the dendritic processes of some of the cells have been lost. The cells of the anterior horns are in a similar condition, but in some of these the chromophilic elements have disappeared from the periphery of the cell and are found nearer the nucleus; in others they persist only at the periphery. Spaces are found in the anterolateral columns from which nerve fibers have disappeared. The vessels of the cord are somewhat sclerotic. The Marchi stain shows intense recent degeneration in the peripheral parts of the posterior columns, in the crossed pyramidal tracts and anterolateral columns, but less degeneration directly in front of the crossed pyramidal tracts and in the median and ventral portions of the posterior columns. The connective tissue of the sciatic nerve and the intima of the vessels in the nerve are proliferated. The nerve fibers of this nerve are normal.

It is evident from the brief abstracts of the foreign representative papers treating of the pathology of pellagra that there is a difference of opinion as regards the systemic or non-systemic appearance of the degeneration in the spinal cord. This is a matter of some importance, as a toxic degeneration is more likely to be non-systemic. In the two cases I have studied the degeneration was diffuse. Indeed, from my own experience I have come to believe that, with the exception of Friedreich's ataxia, there are few disorders causing a truly combined systemic disease of the spinal cord. The degeneration observed in anemia is not systemic, but in the cases I have studied (quite a large number), it has invariably been a diffuse process. It is more intense than that I have observed in pellagra, but not of an essentially different character. The nerve cells, however, are much more affected in pellagra than in anemia.

Writers seem to agree as to the pronounced degenerative changes in the cells of the anterior horns of the spinal cord and of the cortex of the cerebral hemispheres in pellagra. It appears from the first case reported in our paper that the moderate degeneration of the pyraminal tracts predominated clinically over the apparently more intense alteration of the cells of the anterior horns so that exaggeration of patellar reflexes was present.

The view has been expressed (Long) that the thickening of bone about the intervertebral foramina, possibly more in the cervical region, may be a cause of the symptoms. Unfortunately, only the extreme lower part of the cord in the cervical region in Case I was obtained, but posterior and anterior roots from this region, cut separately, showed no distinct degeneration, even by the Marchi stain. Had these roots been implicated in the intervertebral thickening, degeneration would have been detected in them. I have found no indication of root degeneration in the thoracic or lumbosacral region, and the alteration of the posterior columns, at least below the eighth cervical segment, is clearly endogenous, as it is also in the piece of thoracic cord obtained in the second case.

I must conclude, so far as a study of these two cases permits, that pellagra does not always produce, if indeed it ever does produce, a truly systemic disease of the central nervous system; but that the degeneration is caused by some toxic or infectious substance affecting all parts of the cerebrospinal axis, producing cellular degeneration and diffuse degeneration of nerve fibers in the posterior and anterolateral columns. It is not difficult to explain the mental symptoms when cortical degeneration is so intense as may occur in pellagra, and as is seen in the brain I have studied; and the insanity of this disease seems to be of a toxic or infectious character.

The constant involuntary movements of the limbs described by Dr. Anderson, and present also in Dr. Hall's case, are suggestive of the similar movements in strychnine poisoning. In pellagra the degeneration of the cells of the anterior horns probably causing irritation in the muscles innervated by them, the impairment of cerebral inhibition produced by partial degeneration of the pyramidal tracts, and the incoördination produced by the partial degeneration of the posterior columns, the cerebellar tracts, and the cells of Clarke's columns, may all assist in causing involuntary and constant movement. It is sometimes forgotten that holding the hand still and extended is a very perfect form of movement, requiring the degree of tonicity in the flexors and extensors of the limb exactly sufficient to maintain this position.

The spinal pia was considerably thickened in the second case, which was of long duration, but the thickening of the pia was not observed in the first case.

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CEREBRAL RHEUMATISM.

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Through the courtesy of Dr. Spiller, I have been able to report the clinical notes and the microscopical findings in the specimens from three cases of cerebral rheumatism in the laboratory of neuropathology of the University of Pennsylvania; also the clinical history of a fourth case, first under the care of Dr. Barton C. Hirst at the University of Pennsylvania.

The interesting pathological features of these cases, in addition to the meningeal and cellular changes already described by other observers, are the presence of numerous cells similar in every respect to the so called plasma cells, numbers of fatty granular cells, and also an abundance of globular, pale blue staining bodies, probably of albuminous nature, having their origin from the disintegrated tissue.

The great intensity as well as the diffuseness of the cellular changes are exceptional, as is also the vast extent and degree of the neuronophagocytosis.

In the last fifteen or twenty years there has been very little written on the subject of cerebral rheumatism and only a very few cases have been published. This is probably attributable to the effectiveness of the treatment with salicylic acid or its salts, as used at the present time. Such a possibility has been advanced, particularly by Hoppe (1).

From the earlier observers this condition has been reported by Sydenham, Lancisi, Boerhave, Swieten, Stoll, Storck, Barthez, Grierin, Scuda-

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more, Brachet, Granier (2); Vigla (3); Trousseau (4); Girard (5); Jousse (6); Nouet (7); Joffé (8); Rosenthal (9), and many others.

Trousseau divided the cases according to manner of onset, character of symptoms, etc., into six classes, viz., 1, apoplectic; 2, maniacal; 3, meningitic; 4, hydrocephalic; 5, convulsive; 6, choreic. A later classification and what would seem for many cases a more useful and more logical one is that of Joffé; this classification, however, is really restricted to the maniacal and apoplectic forms of Trousseau, and many atypical cases could not be relegated to any of the forms given in this classification. Joffé recognizes three main divisions dependent upon the time and acuteness of onset and duration of the attack. The three forms were spoken of as 1, superacute; 2, acute; 3, subacute.

Atypical cases would require the acceptance of the more complete grouping of Trousseau. Such a difficulty can be seen when one attempts to designate the group to which belong such cases as that of Bramwell (10), where the patient had attacks of cerebral vomiting, headache, fever, and double papillitis, these symptoms following an attack of rheumatism eleven weeks prior to the cerebral outbreak, and healing only under salicylate treatment; or that of Boinet (11), where deep congestion with tumefied, thickened, opalescent, and infiltrated appearance of the meninges were found at autopsy. Another atypical case is that of Martin and Weil (12), where congestion of brain substance and pia mater was present with a large blackish subarachnoid clot at the base, extending through the foramen magnum, and probably causing the rapidly fatal course of the disease in this instance.

Symptoms of cerebral rheumatism, in the most acute cases, corresponding to the so called superacute stage of Joffé, usually occur during the first week of an acute rheumatic fever. A case has been reported where the cerebral symptoms even preceded the other manifestations of the rheumatism, however, the complication may occur at any time during the usual course of the disease, and cases have been observed where the first manifestations were in the final stages or even late during the convalescence. Cases occurring during the late weeks of the malady are generally of a more chronic character, conforming to the subacute type described by Joffé.

The first indications of cerebral involvement are an increase in the body temperature with acceleration of pulse and of respiration and with psychical changes. The patient becomes restless, perhaps depressed, and seems unable to keep his attention fixed upon the surroundings. A delirium, of a low muttering type, will develop, though the patient can be aroused at any time and will be able to converse freely and intelligently. There are generally some slight hallucinations, mostly of an indefinite visual or auditory character. The fever at this stage may be, and usually is, rather high, though many cases reported show no marked increase in temperature. The pulse rate and the respiration are both increased. This stage of restlessness and inquietude may last for several hours or days, or may occur only at night and disappear during the day, retaining this intermittent character for days. After this condition has continued for a variable time, as mentioned, the patient suddenly develops intense fear and anxiety, cries out in terror, and may attempt to leave the bed. The hallucinations are more fixed than in the earlier stage and are those of fear, often of visual and auditory type. At this time, though the joints are still red and greatly swollen, the patient moves the limbs about freely and appears to experience no pain or discomfort. The fever frequently increases at this stage to 104° or 106° F., and in several cases (13) there occurred an additional increase a few minutes before death (from 108.5° to 109.4° F.), and in a case reported by Strumpell (14) there was a post mortem rise to

112° F. At this acute onset the pulse and respiration become greatly increased, the pulse to 120 or even, as in Hoppe's case, 160; the tension and volume varying considerably, as would be expected where such different degrees and kinds of cardiac involvement may be present. The respiration becomes rapid and shallow, and in one case (Hoppe) reached seventy-two a minute. This stage may last for only a fraction of an hour or may extend over several hours or days, the patient then becoming exhausted, as much from the extreme degree of toxæmia as from the effect of the violent exertions during the mania, and passing rapidly into a state of coma, with dilated pupils reacting to light, cyanosed, pale appearance, rapid and feeble pulse, rapid and shallow breathing, which may become even stertorous; in short, the symptoms one would find in any profoundly comatous condition. Cutaneous reflexes are usually diminished and deep reflexes increased. No paralyses of any character occur in clearly cut cases, though the cerebral condition frequently causes incontinence of urine and fæces. *Subsultus tendinum* is usually present. Where meningeal involvement occurs there may be, in addition, the usual signs of meningitis, such as strabismus, Kernig's sign, rigidity of neck, etc. Death ensues rapidly, usually in a few hours or less, or, after ten or twelve hours, the fever recedes, the pulse and respiration become slower, and the patient regains consciousness, and, in the course of a few days, the symptoms disappear. The hallucinations may persist for several days after the patient regains consciousness, but eventually the condition clears up entirely; in a case reported by Joerg (15), the period required before the patient could return to work was almost a year. Labbé (16), Souques (17), Castaigne, and Bell (18) have emphasized the importance of previous or concomitant lesions of the heart, kidney, and liver, believing the fatality in many of the cases to be due to the poor condition of these organs, this being particularly true in cases in which the kidneys are affected; one of the principal channels for ridding the body of toxic products being thus interfered with and causing their undue accumulation. The presence of chronic alcoholism may cause some confusion in the diagnosis, and adds materially to the gravity of the prognosis, particularly in those long standing cases with diseased liver and kidneys, which on account of the chronic gastric disturbance often cannot stand the irritant effect of large doses of salicylates required in the treatment.

Many theories have been advanced as possible causes of this complication, such as toxicity of the cerebrospinal fluid, general toxæmia, hyperpyrexia, hydrocephalic conditions with increased pressure, meningitis, and local invasion of bacilli. In the researches of Poynton and Paine (19), lumps of bacteria (*Diplococci rheumatici*) were found grouped around and in the smaller capillaries, in some cases of chorea of rheumatic origin, and these writers advance the interesting possibility of a toxæmia, more or less local in character, occurring from the toxic products of the broken down tissue at these points.

Josué and Salomon found diplococci in the blood vessels and pia mater in some cases.

Rosenthal, Joffé, and Labbé found the bacillus of Achalme (rheumatic variety) present in the cerebrospinal fluid and blood in cases of this kind, but then cases of severe (20) type have been reported in which, by the most careful examination, no bacteria could be found in any part of the nervous system. Rosenthal and Joffé lay considerable stress on the importance of finding this bacillus, and its proper differentiation from the perfringens variety. Tests made by Souques, Castaigne, and others have demonstrated that the cerebrospinal fluid in these cases of cerebral rheumatism does not have any degree of toxicity above the normal, and this would rather disprove the theory of toxæmia from this source and would favor that of a general toxic condition through the blood or lymph.

Hyperpyrexia as an exciting or causative agent, I think may be safely excluded, as it is only a result of the condition rather than a factor in the development. Many cases have been reported with only a slight fever, often not at all in proportion to the marked degree of the rheumatic toxæmia as evinced by the character of pulse and respiration, etc. In some instances entire absence of fever was noted. Attention should be called to the observations of Poynton and Paine in which they noted, in one case in which only a slight fever was present, that in taking the rectal temperature an increase of 4.7° F. was found. From this statement it would appear that some of the cases reported as having such low fever would possibly have shown a more febrile condition had the temperature been taken *per rectum*.

Congestion of the meninges and even a serous or more severe type of meningitis may be present in some cases, but whether a simple serous meningitis would cause this train of symptoms, or whether they are due to the profound cellular changes, is rather difficult definitely and positively to determine. Hoppe calls attention to this condition, believing that in a disease such as rheumatism, which can produce acute articular swellings and changes in the endocardium and pericardium of such intense character, could very readily cause a similar serous inflammatory condition of the membranes, and this might subsequently recede completely, leaving no traces of its earlier presence, just as the intensely swollen joints become healed with no remaining trace of pathological lesions. The microscopical findings in the cases I am reporting all show marked changes in the cellular elements of the cortex. While some slight differences in the degree of the degenerative changes in the three cases, as well as different parts of the cortex in the same cases, were noticed, the main factors were essentially the same and would lead to the conclusion that the process is essentially a general toxæmia of vascular origin, rather than meningitic or from bacterial invasion. A certain degree of meningitis and œdema was also present, particularly in the third case, but here I think these conditions were only of minor importance. Due to the imperfect methods of staining the cellular elements, the reports of earlier observers show an absence of any determinable lesion, or the presence simply of gross meningeal involvement, but had those earlier cases been examined by the Nissl's method, cell changes similar to those reported by Josué, Salomon, and others would doubtless have been found. It is also possible that the meninges were so extensively involved in the earliest reported cases on account of the futility of the treatment at that period. The ganglion cells as examined by these later writers showed general absence or varying degrees of disintegration of the chromophile bodies. In those instances, in which the cerebral involvement was only of short duration, the cellular changes were not so intense as in those which had extended over a period of days. Only in these latter was there any neuronophagocytosis reported. Josué and Salomon called particular attention to the greater involvement of the frontal and motor regions of the brain in their case.

Cerebral rheumatism must be differentiated from several other conditions and the fact must also be borne in mind that these conditions may also occur in conjunction with cerebral rheumatism. The first and most difficult disease likely to lead to confusion or error in diagnosis is acute alcoholic delirium tremens. An alcoholic history with presence of more terrifying and rapidly changing hallucinations, and particularly the patient's constant seeing of terrifying and moving objects such as snakes, rats, etc. The absence of high fever and other rheumatic signs, also the occurrence at the time typical of a delirium tremens outbreak would easily serve to differentiate these conditions. Salicylate poisoning during the course of rheumatic fever might give a train of symptoms resembling somewhat those of acute cerebral involvement, but they are not accompanied by the fever, increase in pulse and respiration, etc., and the mental changes are usually confined to slight aural or visual hallucinations, and are increased by further doses or disappear when the drug is withheld for a day or so.

Hæmorrhagic encephalitis may at first lead to some confusion, but the early occurrence of focalizing signs, together with the more general character of hallucinations, would enable one to differentiate the conditions. Meningitis should be readily diagnosticated where the more typical symptoms are present, such as stiffness of neck muscles, rigidity of spine, opisthotonos, vomiting, strabismus, etc. At the beginning the two conditions might be mistaken and slight degrees of leptomeningitis could also occur in conjunction with the rheumatic condition.

PROGNOSIS AND TRATMENT.

The prognosis is generally extremely grave, many patients dying in a brief period. The ultimate outcome depends upon the resistant power of the patient, this in turn being subservient to the condition of the various organs of the body, particularly the heart and kidneys. Aside from the symptomatic, no treatment other than that with salicylates has as yet proved to be of any value. Cardiac or kidney insufficiency should be combated by supportive and stimulant measures, as their symptoms would indicate, and excessive temperature be reduced by antipyretics or, preferably, by the use of properly conducted sponging, cold baths, or cold packs. As the onset and course of the cerebral complication is so rapid and so dangerous, the usual method of administration of the salicylates, by mouth, cannot be depended upon. Seibert (21) found that in cases of acute multiarticular rheumatism, where even 180 grains of sodium salicylate had been given daily, by mouth, with no appreciable effect, the use of a twenty per cent. aqueous solution, given hypodermically, in twelve gramme doses, every twelve hours, gave remarkable results. In some instances decided amelioration occurred within three hours of the initial dose; this, therefore, would seem to be a much more active and potent method of administration, particularly where the complication has arisen despite the rather heroic earlier continued use of the drug by mouth. In his earlier cases he found the injection to be very painful, and eventually preceded each dose by a hypodermic injection of one eighth grain cocaine in thirty grammes of water, allowing fifteen minutes to pass for proper anæsthetization, before using the sodium salicylate solution. Where the symptoms of cerebral involvement occur in the severe form, it is doubtful whether any inconvenience or pain would be noticed by the patient, even were the cocaine not given, as such absolute disregard is shown by the patient to the swollen and inflamed joints, so excruciatingly painful before the mental outbreak: therefore the anæsthetic solution could well be dispensed with here. By experimentation an oily suspension was formed, which was less irritable, but could be made only in ten per cent. strength, and consequently was not so efficacious in the acute stage as the aqueous twenty per cent. solution. This oily solution could, however, be used to great advantage after the subsidence of the acute symptoms, and the points in favor of its use at this later period are 1, the presence of camphor as a heart stimulant; 2, the necessity of only one sterilization; 3, the lesser degree of pain and discomfort; 4, slower and more lasting effect. The aqueous solution requires boiling before each injection. The anterolateral parts of the thighs are the best areas for administration, and care should be taken to elevate the skin, so as to inject the fluid into the subcutaneous tissue, as injection into the skin would cause severe necrosis. The syringe should be thoroughly sterilized before each injection. Seibert regulates his dose by giving in the acute cases ten grammes of the twenty per cent. aqueous solution to 100 pounds body weight of patient, this dose being given every twelve hours. He records absence of all toxic symptoms, even with continuous use in large doses by mouth. In the chronic stage he recommends ten grammes of the ten per cent. oily suspension of salicylic acid to each 100 pounds body weight, to be repeated every twenty-four hours. The oily solution should be mixed and boiled and the alcohol then added and the bottle kept air tight to avoid evaporation.

℞	Salicylic acid,	10.00;
	Oil of sesame,	80.00;
	Gum camphor,	5.00;
	Alcohol, pure,	5.00.

M. ft. sol.

Lumbar puncture may be of benefit in some cases, particularly where there are signs of possible supertension of the cerebrospinal fluid. A spinal puncture would also be a great value as a diagnostic measure, particularly in those cases of a doubtful nature.

Case I. Laboratory No. 488. The clinical history and specimen of this case were sent to the laboratory by Dr. N. P. Stauffer.

History: Age, forty-one years. Mother, arthritis deformans (of hands only). One brother died at age of thirty-eight years of pulmonary tuberculosis; otherwise family history negative.

Patient of unusually good health and moral habits. Never smoked or drank. No venereal infection. Of a very kind and retiring disposition. A rather slow but very careful thinker with an excellent memory. Only severe sickness, typhoid at age of eighteen years. Was delirious and illness covered three months time, but recovered with no apparent ill effects. About ten years ago first noticed an inability to start speech. This continued and he consulted various specialists who found no local lesions of throat or nose. He did not stutter nor did he have difficulty with any special words; he simply could not get them started and it became so bad he shunned all society as he was naturally sensitive. About three years before he had an attack of what appeared to be acute inflammatory rheumatism, in which he had swollen and painful feet and hands. He was apparently in good health when he started with his wife on a pleasure trip in July, 1909. After being caught in a rain storm in the mountains, he complained of a sore throat and then painful feet. He was taken to a homœopathic hospital in Denver and was sick for two weeks while they were treating him for acute inflammatory rheumatism. Apparently doing well, delirium suddenly developed during the night, and he had to be restrained. This passed on to coma, and he died five days later without ever having regained consciousness. Dr. Stauffer examined him six hours before death and found a greatly distended abdomen and a failing respiration as the only symptoms. A diagnosis of cerebral meningitis had been made by three homœopaths of Denver. A lumbar puncture was made by the State bacteriologist, Dr. Mitchell, and his examination showed a few staphylococci ,which he said would not warrant him in calling the condition meningitis. *Indol—negative.*

The patient died the same night and a post mortem examination was made. Lungs showed hypostatic congestion both lower lobes, otherwise normal. Heart normal. Liver normal. Spleen normal. Kidneys normal. Pancreas normal. Bladder normal. At junction of ileum to large intestine hæmorrhagic infiltration for eighteen inches was the only lesion we could find. Although about the same distance in the large descending colon a point was almost on verge of ulcerating. Then the skull was opened. No microscopic lesions were found of the membranes or of the outer surface of the brain.

Pathological findings. Sections were made from the paracentral areas, pons, and medulla. In all the slides examined the nerve cells were somewhat swollen and irregular in shape; the staining properties were very poor. Sometimes the protoplasm stained purplish and showed very fine granulation, but it was mostly homogeneous, and no distinct tigroid bodies remained in any of the cells. The nuclei were excentrically placed, nongranular, pale staining, and glistening, and in some cells it was only possible to distinguish the outlines of the nuclei by a very high power. In some instances the nuclei were in small pockets formed by the bulging of the walls of the cell body, producing herniæ. A slight degree of neuronophagocytosis was present throughout the different areas. The large pyramidal cells (giant cells of Betz) showed perhaps more degeneration than the others and were also very greatly pigmented. These cells seem to be more disturbed on the right side while the glial cells stained slightly better on the right side than on the left.

In the cortical sections numerous vacuoles and small, round pale blue staining bodies and a few fatty granular cells were to be seen throughout the field. Along the border of the cortex there was a greenish tinge suggesting the residuum of an old hæmorrhagic process. The meninges were somewhat thickened and infiltrated to the slightest degree with round cells. There seemed to be a greater degree of congestion of the meninges and the intracortical vessels than normal and some degree of circumvascular lymphocytic infiltration on the right side.

The pons and medulla showed the cellular changes to a marked degree, also the blue staining bodies, but in addition, blood pigment with a few red cells was indicative of old hæmorrhages between the pia and the nervous tissue. In the pons several of the smaller vessels were loaded with polymorphonuclear leucocytes, and a few small abscesses occurred throughout the field.

Numerous cells having the typical appearance of plasma cells could be seen in these sections, but all stages of transitional forms to the nerve cell proper could be found, showing that they were only degeneratively altered ganglion cells; then, too, they were entirely too numerous for plasma cells and did not occur in the pia as they should. Amyloid bodies were very numerous, particularly near the periphery. The central canal was greatly widened and flattened, but the contour was not broken. A few cells like *Stäbchen* cells were present but not in the pia. The medulla showed similar changes together with more excessive pigmentation of the cells.

CASE II. Laboratory No. 489. Mrs. ——. Age, forty-five years. White. Admitted to the University Hospital, February 13, 1909, under the care of Dr. Spiller and Dr. Willard. Mother died of tuberculosis, father of paralysis.

Previous personal history: Always well until seven years ago; she was then vaccinated and dated all her trouble from that time.

History of present attack: Began seven years ago in feet and knees, and had gradually affected all the joints of the body. Fingers, elbows, knees were enlarged and painful.

Present condition: Heart and lungs normal; most joints were enlarged and painful; no redness; round some of joints thickening was circumarticular rather than intra-articular. The list of deformities included enlargements and flexion of fingers; enlargement of most joints and flexion of elbows.

February 15th. Seemed more comfortable, less pain and rested better.

February 18th. Continued to improve slightly.

February 22d. Rested well at night, complained of much pain in day time.

February 28th. Very little change noticeable.

March 5th. Slight improvement, seemed to rest better.

March 16th. So much pain as to require morphine to control it.

March 19th. Very bad night, had great deal of pain; seemed suffering especially with legs and arms.

March 20th. Very restless last night, much pain; morphine, one quarter grain.

March 22d. Seemed worse than before, joints very painful.

March 24th. Very delirious at times; vomited early in the evening. Pulse irregular, no dilatation of heart. No audible murmur.

March 27th. This morning had delusion of persecution, thinking that people around her were slandering the character of her parents and were doubting her own morality.

March 28th. Condition seemed about the same. So much distressed that when she talked about her persecution she cried and seemed to be much worried.

March 30th. Seemed somewhat better to-day. Still had delusion.

March 31st. Had idea that some was trying to poison her. Gave no reason why they should. Did not reason out any of her delusions, said they were so because they were so. Had an idea that her son had sold their household belongings and had married a bad woman. She appeared melancholic.

April 6th. Patient was confused, could not give clear answers. Thought that the baking of her joints was for the purpose of putting her off the earth. (Difficult to determine whether she had any tuberculosis.) Delusions did not seem to be systematized. Was said by the nurse in charge to be worse in afternoon as evening came on. Declined to take her medicine, she feared it was poison. Became depressed in afternoon; mental condition was probably toxic, the condition known as cerebral rheumatism. Had been excited and it was necessary to restrain her in bed.

April 11th. Patient's bowels were constipated, enema and cathartics used daily. Her urine showed indican, some phenol, some albumin, and a few casts.

April 18th. Bowel irrigations instituted after cathartics were found to be too severe; patient incontinent and care of her most troublesome.

April 26th. General condition and appetite better, no improvement in mental condition although the patient was quieter and less talkative.

May 12th. Condition about the same. Patient not so lethargic. To-day answered questions intelligibly.

May 22d. Patient was weaker, quieter, and more stuporous. Took nothing but liquids.

May 24th. Condition worse, pulse faster and weaker, temperature 102° F.

May 27th. Four a. m. so very weak could not be raised, pulse very fast.

Died, 6.50 a. m.

Pathological report: Sections were made from anterior and posterior Rolandic, anterior and posterior paracentral, and calcarine areas of the cortex of both sides. In all of the sections neuronophagocytosis, as described by Marinesco (22), was very marked, in some the entire body of the cell being absorbed and leaving only the nucleus surrounded by the mass or clump of phagocytic glial cells. In some instances the cell was in the first stage, with the clustering of the phagocytic cells, and even the tigroid bodies being present. Transitory stages could also be seen throughout. The most intense changes were in the anterior Rolandic and paracentral areas. The ganglion cells were all greatly disturbed, some being even swollen and tumefied with the bulgings occupied by the poorly staining nuclei. The Betz cells in many instances had lost their nuclei entirely and were filled up with yellow pigment, in others the pigment was only slightly increased. This pigment increase, however, extended also to the other cortical regions. The chromatolysis and neuronophagocytosis was marked in all the sections, the nearest approach to normally functionating cells being in the posterior Rolandic zones. The nuclei in the degenerated cells were homogeneous and smaller than normal; the nuclei were larger than normal, eccentrically placed, and were very dark staining. The frontal areas, besides all these changes, showed reduction in the number of ganglion cells with overgrowth of glial cells. The vessels here had some slight degree of congestion with very slight circumvascular lymphocytic infiltration. Numerous cells occurring throughout the nervous tissue and in the pia of all the areas had the characteristics of so called plasma cells. Many of them showed disintegration and vacuolization with appearance of small fatty granular cells. They might be the pseudoplasma cells described by Papadia (23), or broken down plasma cells. They were in too great numbers for plasma cells, and occurred too extensively throughout the tissues. Large, typical fatty granular cells also were present, though in much less numbers. The membranes were slightly congested and thickened, the new connective tissue cells having paler staining and larger nuclei than the older ones, and often looking like the so called *Stäbchen* cells of the German writers; they lacked, however, their strands of protoplasm extending from the poles of the nuclei.

Case III (No. 129). Sent to the university laboratory by Dr. William Egbert Robertson, with a history of acute multiarticular rheumatism of a very severe type. During the second week of the illness the temperature rapidly increased (105° F.) with rapid pulse and respiration, and maniacal outbreak. Patient then became rapidly comatose and died within forty-eight hours of the cerebral involvement. All pain in the joints disappeared with the onset of the mania. Temperature rose to 107° F. shortly before death. Lumbar puncture before death gave negative bacterial result.

Pathology. The sections were taken from the anterior and posterior Rolandic areas of both sides, both paracentral and frontal and occipital (calcarine) areas. Changes were of a character so similar to the two foregoing cases that repetition would be useless. The cellular alterations were not so intense in the frontal areas as in the other portions. The plasmalike cells were only present in the sections of the occipital cortex. Macroscopically the brain appeared very œdematous, this being shown to a marked degree in the microscopical examination.

Case IV (for a time in the service of Dr. B. C. Hirst at the University Hospital—later transferred to Philadelphia Hospital). Mrs. S., white, age thirty-six years; admitted January 1, 1910. Patient brought to Philadelphia Hospital in a confused mental state. Her family physician said that she had an attack of acute articular rheumatism complicating pregnancy. She was delivered spontaneously of a very small (three and one half pounds) infant and in the third day of the puerperium showed signs of mental confusion. This was followed in several days by maniacal outbreaks and she was then transferred to the Philadelphia Hospital. Her mental state would not permit of a definite history being taken at this time—she was comatose and when spoken to would answer incoherently. Had incontinence of urine and fæces.

Physical examination: Poorly nourished. Tongue coated and tremulous. Lungs normal. Abdomen slightly flabby. Fundus of uterus reaching half way to umbilicus—no tenderness. Heart, size apparently normal. Soft, blowing, systolic murmur heard about the fifth interspace to within the anterior axillary line. First aortic sound somewhat roughened, but no thrill palpable. Pulse was soft, compressible, and of moderate tension. Right lower extremity about two inches longer than the left, a condition present since birth. Genitalia: Reddish tinged lochial discharge, small in amount. Both wrists and the right knee were inflamed and swollen.

Condition improved somewhat under salicylate treatment with supportive cardiac measures, the mental stage varying somewhat from time to time. Sometimes she imagined that wires and rags were being pulled out of her body. At others she thought that her baby was not alive, said that "they" had broken its leg and killed it. Imagined that her husband had married some other woman and felt wires and electricity shooting through her body.

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SURGERY OF EXPERIMENTAL LESION OF SPINAL CORD EQUIVALENT TO CRUSH INJURY OF FRACTURE DISLOCATION OF SPINAL COLUMN

A PRELIMINARY REPORT *

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PREFATORY REMARKS

In two former papers I have dwelt at length on the mechanical considerations of injury to the spinal cord in cases of fracture dislocation of the spinal column.[1, 2]

In this communication, therefore, I shall limit myself to the discussion of the experimental work in which I have been engaged for the past year in the laboratories of the University of Pennsylvania.

I would accentuate the fact that this is a preliminary report and that I am fully aware of the amount of control experimentation necessary before an *ex cathedra* statement can be made as to applicability of this work in the human subject.

MEASUREMENT OF IMPACT ON SPINAL CORD IN SPINAL FRACTURE DISLOCATION

In order to arrive at an adequate concept as to the amount of impact a spinal cord could receive and yet recover its function, I designed an instrument whereby a given weight could be dropped from a known height thereby producing a given impact (see the accompanying illustration).

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* From the Department of Experimental Surgery and the Department of Neurology in the University of Pennsylvania. Read at the thirty-seventh annual meeting of the American Neurological Association, Baltimore, May 11-13, 1911.

[1] Allen, A. R.: Injuries of the Spinal Cord, THE JOURNAL A. M. A., March 21, 1908, p. 940.

[2] Am. Jour. Med. Sc., May, 1908.

A dog having been thoroughly anesthetized, a laminectomy is performed, the instrument placed in position and the weight dropped on the spinal cord. The resulting impact in gram-centimeters is the product of the height in centimeters and the weight in grams. The element of friction in the instrument is so inconsiderable that it may be regarded a negligible quantity.

HYPO-IMPACT AND HYPERIMPACT

In my work I have used dogs varying in weight from 7.5 kilos to 18 kilos. The laminectomy has been performed in the lower third of the thoracic region.

I have found that a weight of 30 gm. may be dropped on the spinal cord from a height not exceeding 11.5 cm. with every expectation of an uninterrupted recovery on the part of the animal. The resultant of 339 gram-centimeters is one wholly in accord with complete autorestoration.

So soon as we exceed an impact of 340 gram-centimeters (to speak in round numbers) we obtain symptom pictures of more serious import.

Whereas an impact of 340 gram-centimeters will produce a complete paraplegia of spastic type, which, in course of a week or ten days, will have largely disappeared, so soon as we begin to use impacts of greater magnitude, we find the symptoms more severe and more lasting. Most animals injured by a 420 gram-centimeter impact will present a serious symptom picture; complete spastic paraplegia with scissor-like crossing of the hind limbs, emaciation, paralysis of the bladder and rectum and trophic lesions. An impact of 450 gram-centimeters will produce a symptom picture of complete transverse lesion of the spinal cord and the animal will die in the asthenic and toxic state so familiar to all who have had experience with fracture dislocation of the spinal column in the human subject.

I therefore term all impact below 340 gram-centimeters "hypo-impact," and all impact above this amount "hyperimpact."

THE HYPOTHESIS OF SYMPTOM-COMPLEX OF TRANSVERSE LESION OF THE SPINAL CORD IN FRACTURE DISLOCATION OF THE SPINAL COLUMN

Without restating *in extenso* the views concerning pressure on the cord from fragments of bone or the intramedullary pressure of hemorrhage or edema, with

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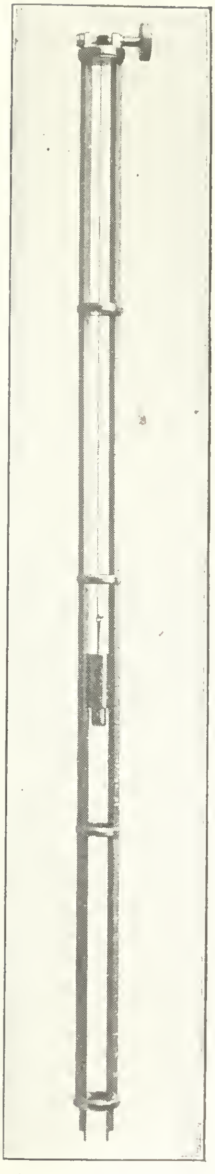

Instrument for producing a measured impact on the spinal cord.

which views I have dealt in previous articles, I would say that I consider it a very uncommon occurrence to find a passive impingement of bone on the spinal cord when the operation of laminectomy is performed for fracture dislocation.

We must assume one of two theories, or probably both, in the etiology of cessation of function in the spinal cord in those cases in which the cord has not been literally cut through: Either there is a destruction of axis cylinders directly consequent to the impact, or else, owing to the impact, there is an edematous and hemorrhagic outpouring into the cord tissue, which by its pressure and chemical activity inhibits temporarily all conduction function, or destroys permanently the spinal cord.

In suggesting a possible treatment to aid and abet an autorestoration, it is obvious that the hypothetical factor of direct injury to the axis cylinder by the impact is beyond our reach, and that we should better confine our attention to the amelioration of the heightened intramedullary pressure.

This has been tried in years past by various means, such as cupping and other antiphlogistic devices. But the amount of benefit obtained has always been open to much doubt and in the fortunate cases, where restoration has taken place, the surgeon has found himself unconsciously imbued with a *post hoc non propter hoc* feeling.

THE SYMPTOM-CONTENT OF LONGITUDINAL SECTION OF THE SPINAL CORD

In order to ascertain the symptom-content of longitudinal section of the spinal cord, I performed laminectomy on a number of dogs. The dura mater was incised in the median line and held apart gently by means of traction sutures. Then with a very fine canaliculus knife longitudinal incisions from 2 to 3 cm. in length were made. In some cases a single incision in the median line, in others, bilateral incisions from the emerging posterior roots forward and inward were made. The incisions were carried completely through the cord. When the dogs came out of ether, they were able to stand and walk, showing very little weakness or awkwardness in walking. There was very little, if any, exaggeration of the reflexes of the hind limbs. A few days sufficed for the animal to recover his normal motor capabilities.

This finding is at variance with the teaching of some of the older physiologists, who have reported paraplegia of spastic type after this procedure.

I cannot but believe that when paralysis ensues after this operation, it is most likely due either to cutting the cord obliquely owing to faulty technic, or else is secondary to inflammatory change from imperfect aseptic precautions. From this work I conclude that median longitudinal section of the spinal cord of the dog is provocative of no symptom of note.

EXPERIMENT OF MEDIAN LONGITUDINAL INCISION IN CASES OF HYPERIMPACT

Given, as the hypothesis, that there is a twofold factor after the impact: (1) the direct injury to axis cylinders from the impact: (2) the outpouring of serum and blood into the substance of the cord; given the condition of the closely investing pia-arachnoid and the inability of drainage; given the comparative absence of symptoms after median longitudinal incision into the cord — the corollary which presented itself was: What effect would a median longitudinal incision into the spinal cord have on subjects submitted to hyperimpact?

The factors suggesting this course were the need for spinal cord drainage, thereby accomplishing a spinal cord decompression, and the fact that the heteromeric neurons in the dog and in man are not of vital importance.

In five dogs I have used a hyperimpact of 540 gram-centimeters (30 gm. at a height of 18 cm.), and then made a median longitudinal incision from 1 to 1.5 cm. in length directly through the impact level and passing altogether through the spinal cord. These dogs made uneventful recoveries, in the worst instances showing only a slight spasticity and awkwardness in the hind limbs after recovery, but not enough to prevent running and jumping.

This impact would have led to the dire consequences which I have pictured above, had not the median longitudinal incision been made. The dogs would have died had not the spinal cord been drained.

DETAILED DESCRIPTION OF OPERATIVE PROCEDURE

An incision of about 6 cm. is made over the spinous processes down to the fascia. The fascia is incised and the muscles pushed well to the side by means of a periosteal elevator. Three spinous processes are cut off by means of bone forceps, and then the lamina of one vertebra is sawn through on each side and the fragment of bone elevated and removed. Further enlargement is obtained by means of specially fashioned rongeur forceps. It is necessary to have at least 3 cm. of the cord exposed so that careful observation can be made. The epidural fat is now wiped away, the impact instrument adjusted and a strong reading-glass focused on the segment of the cord to be struck. The blow is struck, and there is a single jerk of the lower extremities.

The instrument is instantly removed, when it is noted that instead of the cord being the natural color and the dura mater showing as an almost transparent covering through which the small vessels are easily visible, we have now to deal with a dark bluish-purple, swollen mass. I now take a very fine curved needle, and clamping it in a pair of mosquito hemostats I use it as a tenaculum to lift the dura preparatory to incising it centrally. If this incision be carefully made, there at once bulges out of the opening of 2 cm. a dark, beefy-red mass. This is the greatly distended pia-arachnoid. This hyperdistention of the pia-arachnoid extends longitudinally about 4 cm., and when torn by means of a fine tooth forceps it subsides in a great gush of blood and cerebrospinal fluid. It will now be seen that the substance of the cord appears pinker than it should normally.

A median longitudinal incision of at least 1 cm. is made completely through the cord, following which there is a goodly outpouring of blood and serum from the cord substance.

After the active bleeding ceases, the muscles are brought together by a close row of buried Pagenstecher sutures. The fascia is closed by means of a continuous Pagenstecher suture and the skin wound by means of Michel clamps. The dura mater in the dog is too tight to warrant trying to suture it, as would most certainly be indicated in man.

The dog is completely paraplegic following the operation, and it may be three or four days before there is any indication of return of power. In a week's time he is standing and moving about his cage with a very spastic gait. Little by little the power returns and the spasticity diminishes. The end-result is a complete restoration of motor power. The animal is able to run and jump and has complete control of bladder and rectum.

The only point in the convalescence which may give trouble is the development of what I might term an artificial spina bifida. Owing to the difficulty of closing the dura mater in the dog, there is a leakage of cerebrospinal fluid upward forming a fluctuating tumescence, possibly the size of an egg, at the seat of operation. This it is my practice to evacuate by means of an aspirating needle under the most stringent aseptic precautions.

This experimental work has been carried out under full ether or cloroform anesthesia; and at no time have the dogs given evidence of suffering pain.

GENERAL CONSIDERATIONS: OUTLINE OF FUTURE WORK IN THIS FIELD

There are many steps between the results above mentioned and the adoption of the median longitudinal incision through the injured area as a desirable method of procedure in cases of spinal cord injury in fracture dislocation in the human subject.

The first important step to determine is: How long after the injury to the dog's spinal cord can one wait and still obtain the good effect of the drainage from median longitudinal incision?

I shall conduct a series of cases of hyperimpact in which the median longitudinal incision is made two, four, six and twelve hours after the impact; then a series in which a period of twenty-four hours elapses before the cord is cut.

I have already tried to aid the drainage by applying a Bier stasis-hyperemia cup over the laminectomy area the day after the impact operation of median longitudinal incision had been performed. This I applied for half an hour twice a day, but the result was most undesirable. The dog showed every symptom of complete transverse lesion shortly after the first treatment and died about four days later. On histologic examination, I found the gray matter of the cord greatly softened and hemorrhagic for a distance of 2 cm. above and 2 cm. below the impact area. It looked just as if the gray matter, naturally lacking in *Widerstandsfähigkeit* (resisting power) had given way to the negative pressure.

Of course, this is only one case in which the Bier's method was used. It is altogether possible that, with a more gentle suction, a better or favorable result might have been obtained.

I wish to call attention to a point which has impressed me greatly in all this work. I refer to the effect of severe hemorrhage during the operation on the period of convalescence.

Hitzig[3] has shown the baneful effect of exsanguination on the central nervous system. I also have noted in a previous work the great change in conductivity in the lateral columns of the spinal cord in animals (dogs) slowly exsanguinated.[4] There is no doubt to my mind that whenever I had much hemorrhage, the animal's convalescence was prolonged, his spinal cord symptoms more severe than the impact would warrant and the chances of an infection greatly augmented. I am convinced that this is an important element in the failure of operations on the brain and cord.

It is not my purpose to take up the histopathology of this work until a future communication. I shall only say that I have been surprised at the amount of ascending and descending degeneration that I have found by the Marchi method in the spinal cords of dogs which had recovered their motor power completely and which I had killed in order to make the histopathologic study.

My tentative conclusion is that in cases of fracture dislocation of the spinal column in the human subject, in which there exists the symptom picture of transverse lesion of the spinal cord, it were well to perform the operation of laminectomy at the earliest possible moment, and if the cord be not completely severed to make a median longitudinal incision through the area of impact by means of a fine canaliculus knife in order to drain the injured tissue of the products of edema and hemorrhage.

2013 Spruce Street.

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[3] Hitzig: Physiologische und klinische Untersuchungen über das Gehirn, Berlin, 1904, part 1, p. 23.

[4] Allen, A. R.: Hemorrhage Into the Ventricles, THE JOURNAL A. M. A., July 18, 1908, p. 216.

DR. SPILLER'S ADDRESS TO MEDICAL STUDENTS

Delivered at the Opening of the Medical School

Owing to the delay in publication and the pressure of new matter we are compelled to omit the details of the opening of the departments. Following we have the pleasure of presenting the address of Dr. William Gibson Spiller, Professor of Neuro-Pathology and Associate Professor of Neurology at the Medical School. Dr. Spiller said:

The oldest medical school of the United States begins its one hundredth and forty-sixth session. A speaker may well hesitate in selecting the subject of his remarks to such a gathering of students from near and far. The temptation meets him to dwell upon the history of this ancient institution, to describe how the University had its beginning, and to picture its triumphal progress through the long period of years. All this has been presented in so much better form than I could hope to do it that it has seemed wiser to commence my remarks with some reference to the recent development of medical education throughout this country.

The changes that have been taking place in medical education during the past few years have been marvelous. It has not been long since the medical student was obliged to rely on didactic teaching, and had little or no intimate contact with the patient. He was allowed, possibly at infrequent intervals, in a large group of men, to listen to a heart murmur, and often persuaded himself that he had heard what possibly he had not. Wards were in large measure closed to students. This was the state of affairs in our best medical schools. The country was cursed with a large number of schools graduating badly prepared men. It was customary, indeed almost obligatory, for a medical graduate seeking a more thorough education to go to Europe after receiving his degree. There he found that in groups of about eight men he might study disease as presented by the patient rather than by the textbook or the lecturer. Other advantages were obtained in the mingling with men of a different training, in observing new customs, in becoming familiar with the German or French language. A foreign sojourn, while no longer necessary, is still advisable for those who are able to accomplish it. The acquirement of the German or French language is in itself an invaluable asset.

The American student sometimes felt obliged to blush for his country, for our students did not always make a creditable showing. On the whole, from my own observation, those who sought foreign advantages were earnest and eager to accept the opportunities offered.

The Council on Medical Education of the American Medical Association, with the Carnegie Foundation for the Advancement of Teaching and other agencies, has done much to raise our standards. During 1906 and 1907 the Council made the first personal tour of inspection of the medical colleges of the United States that had ever been made. This inspection revealed the fact that nearly a third of the medical schools existing at that time were seriously defective in their methods, standards and equipment. The amount of money given for medical education has increased from a few thousands of dollars during 1904 to several millions of dollars during the last year.

The vast improvement in the "Journal of the American Medical Association" under the present editorship has done much to educate physicians throughout the country. This journal has now become a source of information that no physician can ignore without serious loss to himself.

Thirteen medical colleges have been closed during the past year; eight of these were suspended and five were merged with other institutions. During the past seven years sixty-four medical colleges have ceased to exist either through merger or otherwise. During the same time eighteen new colleges were organized. The total number of medical students (matriculants) in the United States for the year ending June 30, 1911, excluding special students, was 19,786, a decrease of 1740 below 1910, of 8356 below 1904, when the highest number of students was enrolled.

The total number of graduates for the year ending June 30, 1911, was 4273, a decrease of 167 below 1910 and of 707 below 1907. The total this year is 1474 less than in 1904, when there were 5747 graduates.

The requirement for admission of one or two years of collegiate work has been adopted during the past year by twenty-two of the better class medical schools of the United States, making a total of forty-one which are now enforcing the higher standard.

The number of physicians in Germany, more especially in Bohemia, has become so large that active measures have been taken to deter prospective students from pursuing the medical course. At a recent meeting of German physicians in Bohemia a resolution was passed that the students about to enter the university should be warned against turning to medicine. They should be informed that within the last five years the number of medical students in that country has risen from 2509 to 5319, that is, 112 per cent., while the number of all university students has increased by barely 40 per cent. They should also consider that fully one-third of all practitioners in Bohemia earn less than $400 a year, and that the number of doctors has increased in the last two decades by 76 per cent., while the population has increased only by 19 per cent.

There seems to be a scarcity of physicians in Russia. European Russia has only one physician to 8000 inhabitants, France, one to 2630, England, one to 1730, and the United States, one to 650. In view of these facts, the necessity of raising our standard of medical education and of preventing the yearly turning out of half-ripe physicians is apparent.

Some words recently used by Dr. Ira Remsen, the President of Johns Hopkins University, regarding university aims are pregnant with meaning. "It is easy to show," he says, "that intellectual advancement is dependent upon research. Further industrial progress is directly connected with intellectual advancement, so that the welfare of a nation is closely related to the cultivation of research." And again: "A dozen good strong universities properly distributed would amply supply the needs of the country, and better work would be done by concentrating it in a few places. A dozen is probably a large estimate."

The University of Pennsylvania has always stood in the front rank of American institutions of learning, and if it is to maintain this position it is necessary, in view of the advancement of medicine throughout the country and in accordance with the desire for research that is steadily growing, that the entrance requirements of the medical school should be high. It is for these reasons that the entering class is small, but the motto has become "Not many but much." The Dean has informed me that were our requirements the same as those of a few years back we should have a very large First-Year Class. Medicine must have high aims.

United States Senator Owen, of Oklahoma, recently referred to the charge that the medical profession is forming a trust under the lead of the American Medical Association, in abolishing harmful nostrums. "They would be engaged," he said, "in tearing down their own business; they would be engaged in depriving themselves of their patients from whom they make their living. It would be the only trust in existence which is concerned in diminishing its own revenues and destroying its own financial foundation. Such a trust as that is a very noble trust and one that deserves encouragement."

Words such as these are encouraging. We are so accustomed to the threadbare jokes of the comic papers in relation to the doctor, who shares with the mother-in-law as a proper target for the shafts of pseudo-witticism, that we are pleased to find a proper appreciation in the public mind of our profession. Physicians labor under the disadvantage that they are consulted only by necessity. One does not seek sickness, and when he is forced thereby to consult a physician he sometimes transfers to him the onus of the illness. It is true that physicians are continually striving to prevent rather than cure sickness, but success is not so great as is sometimes represented, thus Maeterlinck, in his play, "The Blue Bird," in which he endows inanimate things with body and spirit, speaks of the sicknesses as being very quiet and unhappy, because man, for some time, has been waging such a determined war upon them. The doctors have been so unkind to them. One, the smallest, Cold-in-the-Head, is frisky and escapes, but because winter has not yet come he is driven back and the door is shut.

The holding before the living the names of the illustrious dead is a stimulus to ambition. As a people we are too much inclined to believe that men have their day and cease to be. I have seen the funeral honors paid to Billroth, Pasteur and Tennyson, but I have never seen the consignment to the tomb of a man illustrious in literature, science or the arts in this country with such honors as were shown to these three men. It cannot be said that we are incapable of appreciating intellectual triumphs. We have been, and are becoming still more, originators in medicine, but we are copyists in many things. The design of the tomb of our great general, Grant, is borrowed in its interior from that of Napoleon in the Hôtel des Invalides in Paris, and Baedeker, that constant guide of the tourist abroad, speaks of the monument as somewhat disappointing in its general proportions and effect, but as much more impressive in its interior. We may reject this verdict if we dare, but if we do we shall be braver than was the old Emperor William I, of Germany, of whom it is related that once in conversation with Bismarck he excused himself, remarking that Baedeker had stated that he, the Emperor, always appeared at a certain window of the palace at that hour to be greeted by the people, and he must obey Baedeker's directions.

We show some absurdities in our hero worship. A more incongruous collection of statuary than that in the National Capitol would be hard to find. We establish a Hall of Fame and an Edgar Allen Poe must wait for recognition. We have now and then an Edwin Austin Abbey who as the decorator of a state capitol, or a public library teaches us the majestic and original in art. The victories of mind are achievements we should be proud of. The engraving of the names of the great on our buildings devoted to learning is a pleasing custom. The University of Pennsylvania has not been backward in this respect. On the Law Building one may read the names of many who have added lustre to the legal profession. The new building of zoology is similarly adorned, and it is a cause for regret that the custom was not followed in building the Medical Laboratories. The student might find names with which he was not familiar, and would be inspired to study the lives of those who being dead yet speak. The College of Physicians of Philadelphia in its new building has honored physicians by inscribing their names upon its walls. In language from a most thrilling dedicatory address we may say: "From these honored dead we take increased devotion to that cause for which they gave the last full measure of devotion." The quotation is apt, for a life spent in the alleviation of suffering and sickness, and it may be, ended by disease incurred in the discharge of duty, is not less noble than one terminated on the battlefield.

The question of the advisability of choosing a specialty will be presented to some of you. It is not well for an undergraduate student to select a specialty unless circumstances be unusual. It is desirable that he first become well informed in general medicine, and few who have not been some years out of the medical school know for what they are best fitted and what appeals to them most. Many a man has determined that he would follow a certain branch of medicine and circumstances have forced him to change his course. Indeed some men are embryo specialists successively in several branches before they find their proper place. "Blest is the man," says Carlyle, "who has found his work. Let him ask no other blessedness." How difficult it is to find one's work. Weir Mitchell has touched charmingly on this subject in his recent novel, "John Sherwood, Ironmaster," in which his hero enters on his real life at the age of thirty-five, having had his better self buried until illness at that period opened a new existence to him.

Occasionally a genius of the first rank has given little indication of his special talent in his early life. No one thought that the boy, Richard Wagner, was especially musical. While Beethoven wept over his early musical lessons he yet showed ability. Wagner, on the other hand, at the age of eight years was regarded by his stepfather as having some talent for music, but of this he was not certain. His music teacher could not be convinced of the existence of musical talent in the boy. Wagner early wrote poetry, and arranged a tragedy in which he killed forty-two persons, and revived some of these in the form of ghosts to bring his play to a finish. An overture composed in early manhood produced excessive laughter from the musicians who attempted to perform it, and the audience roared in sympathy.

While it seems unwise to suggest that an undergraduate student should choose a specialty, it may not seem out of place to you if I sketch in something of the style of the impressionistic school a little of the development of that specialty, neurology, with which I am more familiar, especially as represented by its greatest exponent. It is a branch of medicine which has to do with the highest development of the body and which in America has not yet had its relatively important position. No man in general practice can ignore the disorders of the nervous system, and the manifestations of functional derangement form a large part of disease.

Neurology has close connections with all branches of medicine, but it should be regarded only in its relative importance.

This country owes a debt of gratitude to Weir Mitchell, the Seguins, the elder Hammond and our own Professor of Neurology among others, but it is in France above all other countries that neurology had its chief growth under Charcot. Few names in medical history shine with brighter lustre than that of Charcot, and to describe his life is to describe the development of neurology in France and indirectly in other countries. The Germans have contributed greatly to neurological advancement, and the names of Romberg, Friedreich, v. Leyden, Erb and, more recently, Oppenheim, are those to conjure with, and yet during the lifetime of Charcot it was not uncommon for a German neurologist to worship at the French shrine. This is all the more remarkable in that neurology has been a branch of medicine which has not failed to interest German physicians. The National Society of German Neurologists at its fourth annual meeting last fall had on its membership roll 403 active members; the American Neurological Association at its thirty-seventh annual meeting last spring had on its roll 112 active members. It is difficult to understand why neurology has not yet come into its own in our country. Whether it be that it demands a thoroughness to detail which has not as yet appealed strongly to us on account of our love of activity, I know not. We are restless. The laborious German Arbeit demands too much time, we must be "practical." Our restlessness is evident in all branches of life, in our play as well as in our work. We are scarcely satisfied in traveling a mile a minute, but must do more to annihilate time and space. As Weir Mitchell puts it: "He is passing, the sensitive gourmet of literature. We are gourmands and gorge ourselves with crude food at the newspaper trough or the magazine lunch counter."

If this be true of general literature, what may be said of some of the medical writing in which verbosity, superficiality and carelessness are glaring faults.

Charcot was in a measure neurology personified. To one who has not lived in the atmosphere of French medicine it is difficult to appreciate how he dominated the Parisian school.

He was a distinguished physician in general medicine before he became the greatest of the French neurologists. There was, I think, but one other man among the French for whom this claim could be disputed, viz.: Duchenne, of Boulogne. His work on muscular atrophy, electricity as applied to the human body, bulbar palsy, tabes dorsalis, etc., has justly placed him among the most distinguished physicians of all time.

Jean Martin Charcot was born in Paris, November 29, 1825. His father was a carriage builder, but, recognizing that his son Jean gave promise of great things, he bestowed upon him a better education than he himself had received. Jean hesitated in his choice between the pursuit of art and that of medicine, and finally selected the latter, although his artistic tastes were never buried. The following anecdote is told concerning the Charcot family. The father called his boys about him and said: "You are all old enough to choose professions. My means are not sufficient for further education of you all, although they will allow me to give advantages to one of you. I have determined that you, Martin, shall be a carriage builder as I am. You, Emile, will enter the army; and your brother Eugene will be a sailor. As for you, Jean, as you have much ability in drawing, you may be a painter, if you wish, but as you are diligent you may complete your studies and become a physician."

In 1848, when twenty-three years of age, Charcot became an interne in the hospitals of Paris, and served one year at the Salpêtrière, a hospital which later was to owe its chief glory to the learning of this man. He graduated in medicine in 1853. From 1853 to 1855 he was chief of the medical clinic, and in 1862 was admitted to the medical faculty and became physician to the Salpêtrière. His medical career thus began and ended in the Paris poorhouse for old women, a place in some respects resembling our own Blockley. Charcot had the power of observation in a degree few men possess. One of his favorite sayings was to the effect that we see only those things we have once been taught to see. He combined clinical and pathological investigation in a most successful manner. From 1872 to 1881 he was professor of pathology, and he gave lectures on diseases of the different viscera of the human body.

The French system sometimes works in a very peculiar way. When a vacancy occurs the man who stands in the line of promotion is likely to obtain the chair, whether he be especially fitted for it or not. Thus Dejerine, who is distinguished as a neurologist, accepted the chair of internal pathology, and has recently resigned it for that of neurology, for which he was especially adapted. Maurice Letulle, who is known in pathologic anatomy, has been appointed to the chair of history of medicine. Two years ago the chair of pathologic anatomy became vacant and the place was filled by one who had distinguished himself in neurology, Pierre Marie. Thus a distinguished neurologist is teaching pathologic anatomy and a pathologic-anatomist, instead of teaching pathologic anatomy, is about to give a course in the history of medicine.

In 1882 the professorship of neurology was created especially for Charcot, and from this time dates the fame of the Salpêtrière as the greatest neurological hospital in the world. One almost wonders why the French, with their love for hero worship, have not changed the name of this building to the Hospital Charcot. From no other place came within so few years such a group of brilliant neurologists. The name of Charcot drew men from every nation to Paris, and his lecture room, which was large, was always well filled. This room was peculiar, as was almost everything belonging to Charcot. It was of great length and not so broad as long, and the floor descended gradually from the back toward the platform, which extended across the entire width of the lecture room, and was raised about a foot or two above the floor. The platform was sufficiently large to accommodate fifty or more persons, the professor, the assistants, the guests and the patients.

Charcot lectured twice weekly. The Tuesday meeting was a presentation of the most interesting nervous cases that were to be found in the outpatient department, but the Friday discourse was a carefully prepared paper that was always worthy of publication in a medical journal of the highest rank.

Associated with Charcot in his early medical career was Vulpian, among whose most illustrious pupils were Raymond, Charcot's immediate successor, and Dejerine, the present incumbent of the chair. Raymond had the rare fortune to call both Charcot and Vulpian his teachers.

When Charcot began his medical studies almost nothing was known concerning localization. Fritsch, Hitzig and Ferrier had not done their work, and the views of Flourens, that the cerebrum is a homogeneous mass and that symptoms result proportional to the amount of brain tissue lost, received the sanction of scientists. Broca's discovery of the location of the motor speech center was made in 1863. The discovery of the excitability of the cerebral cortex to electricity was of the greatest aid in the study of cerebral localization, although at first it was accepted with much skepticism. Charcot was among the earliest to teach cerebral localization. We are so familiar with the facts gained within the last twenty-five years that possibly we do not fully realize what a revolution in medicine cerebral localization has made. Surgery of the brain has become possible by this knowledge. Charcot's method of determining the situation of cerebral centers was to seek in pathological conditions the cause of the symptoms he had observed during the life of the patient. Among the most distinguished of Charcot's opponents in these early days of cerebral localization was Brown Séquard, at one time professor at Harvard, who offered the results obtained by himself experimentally as contradictory to Charcot's teaching. It is to Charcot that we owe much of our knowledge regarding the motor region of the cortex.

Although it was Türck who first described secondary degeneration, it was Charcot who showed that degeneration of motor tracts would result from a lesion of the spinal cord or motor cortex of the brain as truly as from a lesion of the internal capsule mentioned by Türck. It was he who first taught the trophic influence of the motor cells of the anterior horns of the spinal cord. It was he who foretold that in acute and chronic poliomyelitis the nerve cells of the anterior horns of the spinal cord would be found diseased. He, in association with Bouchard, taught that cerebral hemorrhage was the result of rupture of miliary aneurysms. He taught us to recognize pseudo-bulbar palsy.

To him we owe much of our knowledge concerning paralysis agitans, multiple sclerosis, tabes, amyotrophic lateral sclerosis, which the French love to call Charcot's disease; lateral sclerosis, hysteria, ophthalmic and ophthalmoplegic migraine, the Charcot-Marie form of muscular atrophy, and many other affections of the nervous system. His name was on the editorial board of many of the French medical journals, and it has also been used in the designation of many phenomena of disease.

In 1859 Charcot showed that intermittent claudication in horse or man was caused by ischemia. This peculiar affection, which many years later was called by Erb intermittent lameness, was still almost unrecognized by the medical profession thirty years after Charcot first called attention to it, and indeed it was not until Erb made it the subject of a monograph some years ago that it began to excite widespread interest. A number of cases have been reported in America.

The name Salpêtrière refers to saltpeter, which at one time was manufactured on the site of the hospital. The grounds of the hospital are extensive and the buildings very old. The beds for the patients are old-fashioned and stand high from the floor, and about some of them are the curtains such as our ancestors used. The nurses are not like those found in the American and English hospitals and come from the lower walks of life. Many of the buildings are low, about two stories high. They are made of stone. Blockley in comparison with the Salpêtrière is a modern hospital.

The grounds of the Salpêtrière are more beautiful than those of Blockley, and the buildings are not so crowded. There are some fine old trees under which the afflicted inmates can spend the summer days. The number of beds at the Salpêtrière is about the same as at Blockley.

Charcot was very active until his sudden death at the age of sixty-seven years. He died August 16, 1893. He is now but a memory at the Salpêtrière, but a memory that will never fade so long as the study of medicine lasts.

I may translate a few of the words written by Erb, one of Germany's greatest physicians, immediately after the death of Charcot:

"A few weeks ago the surprising and distressing news came to us from France: Charcot is dead. He died suddenly from an attack of angina pectoris, far from his family, in the arms of two of his pupils and friends while he was on a journey for pleasure. The brightest star in the medical heaven of France has faded; Paris and all France have suffered a heavy, an irreparable loss, which is so much greater because it was entirely unexpected."

In bringing this address to a close let me express the hope that your years spent at the University of Pennsylvania may be among the most fruitful of your lives. May it be your desire to add to medical knowledge as well as to absorb a little of that already acquired.

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Diagnosis and Pathology of Tumors of the Pons. A Pathological Report of One Case.

By JOHN. H. W. RHEIN, M.D.,
Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College for Graduates in Medicine, Neurologist to the Howard Hospital, Physician
to the Philadelphia Home for Incurables, Philadelphia.

(Read before the Section on Medicine, Medical Society of the State of Pennsylvania, Harrisburg Session, September 28, 1911.)

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Diagnosis and Pathology of Tumors of the Pons. A Pathological Report of One Case.*

BY JOHN H. W. RHEIN, M.D.,
Philadelphia.

The following case is interesting as illustrating some of the difficulties met with in the diagnosis of tumor of the pons. It also shows how extensive gliomas may become without causing clinical manifestations sufficient to allow the true size or extent of the growth to be suspected.

The patient, E. W., aged six, presented negative family history. She had enjoyed good health until May, 1909, when her adenoids were removed. In the early summer she had fallen backward striking the occiput but causing no serious symptoms. During July and August of the same year she suffered from frequent attacks of headaches and later in September she began to have attacks of vomiting. On October 11, there was a slight rise of temperature to 100° and she complained of rheumatic pains which were associated with a slight endocarditis. Two weeks later weakness of the right side of the face was noted.

The patient having recovered from endocarditis, I was consulted December 5, 1909, on account of slight persistent weakness of the legs and some weakness of the right side of the face. There had been frequent vomiting of mucus in the morning, since October 11, 1909, without apparent cause, the patient being able to eat immediately afterwards. There had been no headaches however since summer.

Upon examination I found that the gait was feeble and that she seemed to favor the left leg in walking. There was however, at this time, no true paralysis of the extremities. The face on the right side was paretic. She could not close the right eye so promptly as the left and the right side of the mouth drooped slightly. The tongue was protruded to the left. Some transient weakness of the left external rectus muscle had been observed by the parents on one occasion.

On December 18, it was noted that suddenly the left arm and leg became distinctly weak. The left arm was used in an ataxic manner, the gait was somewhat hemiplegic, and sensation to the pin-point was lowered on the left side of the face at this examination. Both knee jerks were increased and equal, and ankle clonus and the Babinski phenomenon were present on the left side. The pupils were equal and reacted to light and in accommodation. Shortly after this period there was on two occasions retention of urine so that the bladder became distended.

On January 15, 1910, Dr. Posey noted some slight ptosis of both sides in addition to the paralysis of the external rectus on the right. There were no eye-ground changes.

On January 20, there was sudden loss of

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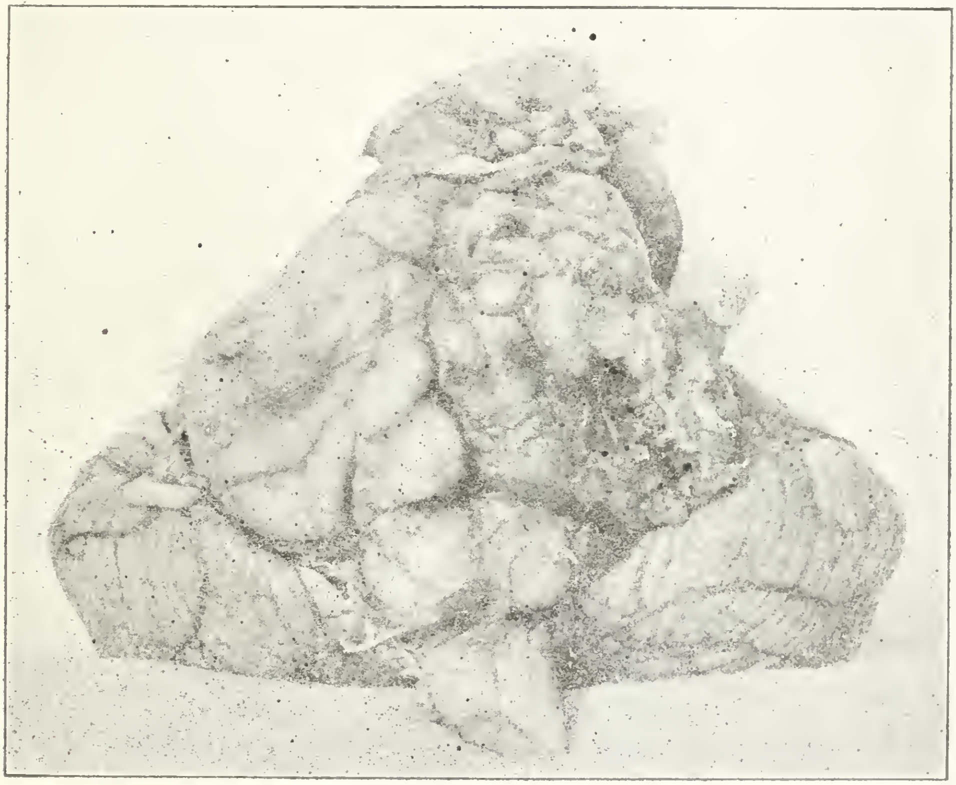
*From the Department of Neurology and the Laboratory of Neuropathology at the University of Pennsylvania speech which lasted for three quarters of an hour and this was followed by an attack of vomiting. At this time weakness of the right external rectus muscle was observed and the palsy of the left arm and leg became almost complete. The patient became irritable and her speech was indistinct. At the same time, however, the sensorium remained unusually clear. Sensation of the legs and arms was apparently intact to the pin prick. In view of the autopsy findings this absence of sensory disturbance is remarkable.



Shows enlarged pons and area of softening on the left side.

On February 2, she had not talked for nine days, and had difficulty in swallowing. The patient at this time lay almost motionless, eating but little. The temperature ranged from 99° to 101°. Ankle clonus and Babinski phenomenon on both sides with increased bilateral knee jerks were observed. From this date until March 9, when the patient died, the condition was as follows: There was paralysis of both arms and legs with slight general spasticity. Complete anarthria was present and she was unable to protrude the tongue beyond the line of the teeth. Both sides of the face were motionless. There was paralysis of the external rectus on both sides. Associated upward movements of the eyeballs were possible but associated movements downward and laterally were paralyzed. There was no apparent deafness and the eyesight appeared to be good until the last. The sensorium was clear until within a short time before death. During the last few days there were a few general convulsive attacks.

At the autopsy the entire pons and upper part of the medulla oblongata were found to be much enlarged, evidently due to an infiltrating glioma. The left side of the pons was softened and hemorrhagic. The tumor did not extend into the peduncle but invaded the upper portion of the medulla. A microscopic study of the tumor confirmed the diagnosis of glioma.

In making a diagnosis of tumors of the pons, one must be familiar with the anatomy of this region. It must be borne in mind that, besides the pyramidal tracts, the pons lodges the nuclei of the fourth, fifth, sixth and seventh nerves. It must also be borne in mind that the superficial transverse fibers contain chiefly cerebellar fasciculi; that the deep transverse fibers, namely the trapezium connecting the superior olives of the same and opposite sides, contain cochlear fibers. The superior olive is believed to be the seat of the function by which the head and eyes are turned towards the direction of auditory impressions and to connect with both of the accessory nuclei of the eighth nerve. They connect also with one or possibly both cerebellar roof nuclei, with nuclei of the sixth on the same side, with the dorsal longitudinal fasciculus of the opposite side, and with the lateral columns of the cord and with the postgeminum. (Mills.[1])

In the preoblongata are also the dorsal longitudinal bundle and the central tegmental tract. (Mills.)

In the lemniscus, or fillet, we have the sensory tract, containing fibers which transmit pain and temperature sense, and also the acoustic tract. In the median portions of the pyramidal tracts we find also the motor speech pathways.

It must also be remembered that a lesion in the lower third of the pons after the decussation of the seventh nerve will give rise to a paralysis of the facial nerve on one side and paralysis of the body on the opposite side, providing the pyramidal tract is implicated. If the fifth nerve is involved, there develops anesthesia on one side of the face and paralysis of the body on the opposite side. If the sixth nerve is the seat of the lesion, which at the same time implicates the pyramidal tracts, there results a conjugate deviation of the eyes towards the side of the lesion and away from the paralyzed limbs. If the sensory fibers are implicated there is loss of sensation on one side and paralysis on the opposite. If the middle cerebellar peduncle be implicated it gives rise to vertigo, vomiting, and tinnitus, or there may be forced movements or forced positions.

A lesion of the pons in the region of the aqueduct of Sylvius, implicating the posterior longitudinal bundle, would cause paralysis of associated lateral movements of the eyeballs. (Spiller.[2]) Paralysis of associated upward or downward movements of the eyeballs would indicate a lesion in the vicinity of the ocular motor nucleus. (Spiller.)

With this brief résumé of the functions and anatomy of the pons, I will comment more or less briefly upon some of the symptoms and other data obtained from a study of forty-five cases of tumor of the pons with autopsy taken from literature, including the case reported in this paper.

The usual duration of the symptoms before the fatal termination is four or five months, though there are on record cases in which the patients have died after symptoms have lasted but for a day, and there was one patient who died at the expiration of two and one half years. Of 27 cases in which the duration of the disease was mentioned, 21 of the patients died within a year after the first symptoms.

A study of the age of the patients shows nothing very definite, except perhaps that many of these cases occur in children under ten years of age. Of 42 cases in which the age was mentioned there were 14 under 10 years of age, 5 between 10 and 20 years, 5 between 20 and 30 years, 8 between 30 and 40 years, 5 between 40 and 50 years, 4 between 50 and 60 years, and 1 was over 60 years of age. Of this number 33 were in males and 9 in females.

A history of injury was obtained in only 7 cases, consisting of blows on the head, or of falls which resulted in the head being struck. There was, in the case reported in this paper, a history of a fall in which the back of the head was struck but without unpleasant sequelæ.

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[1] Mills: The Nervous System and Its Diseases, 1898.

[2] Spiller: *Jour. Nerv. and Ment. Dis.*, 1905, July and Aug.

Studying the nature of the first symptom there is nothing very characteristic obtained from these statistics. Headache, vertigo, or vomiting occurred in 33 cases in which mention was made of the first symptom. Some peripheral cranial palsy, consisting of double vision, facial paralysis, implication of the fifth nerve, or incoordination, occurred early in the history of many of these cases. Vertigo was present in 20 cases. Headache, an early and persistent symptom of brain tumors, was mentioned in 27 cases. Ladame[3] found it present in 16 of 26 cases collected from the literature. The situation of pain seemed to have no significance, for it occurred with equal frequency in all parts of the cranium. This is the experience of most authorities. Vomiting, a common symptom in brain tumors is frequently met with in tumor of the pons. It was mentioned as being present in 23 of the cases. Bernhardt,[4] however, considered this a rare symptom, though he found it in 11 out of 19 cases.

Convulsions, which are present in over 50 per cent. of brain tumors, do not seem to be so frequent in tumors of the pons, and were present in only 7 cases. They have been noted infrequently by Nothnagel,[5] Bernhardt, and Ladame.

Choked disk, which according to most authorities occurs in about 80 per cent. of the cases of brain tumors in general, is uncommon in tumors of the pons. It was mentioned in only 10 cases, although blurring of the disk occurred in one case, the disk was opaque and hyperemic in another case, the disks were hazy in a third case and in a fourth case it was edematous. Rarity of the occurrence of optic neuritis is emphasized by other authorities, notably Bernhardt.

Mental impairment occurs quite commonly in the final stages of brain tumors, and a study of the cases in my tables shows that this symptom obtains frequently in pontine neoplasms. It is not invariably present however, as in the case reported in this paper the sensorium was perfectly clear until a short time before death.

In the cases collected coma was present four times, semicoma or stupor eight times, somnolence twice, dulling of the mental faculties five times, and the memory was poor or lost in three cases.

The localizing symptoms of tumor of the pons will now be taken into consideration. Of these the most characteristic is the Gubler-Millard type of hemiplegia, consisting of paralysis of the face on one side and paralysis of the limbs on the opposite side, alone or with implication of one or more of the other cranial nerves arising in the pons or its neighborhood. While it is usual to observe crossed hemiplegia confined to one or more cranial nerves on one side and paralysis of the limbs on the opposite side, this is found during the early stages of the disease and sooner or later gives way to a confused symptom complex in which will be found cranial nerve palsies also on the same side as the hemiplegia and general palsies. The Gubler-Millard type of paralysis is an early symptom in most instances and is not a persistently uncomplicated one.

In the case reported in this paper, an early symptom was paresis of the right side of the face and weakness of the left arm and leg. This, however, was preceded by a transient palsy of the left sixth nerve, which confused the significance of the Gubler-Millard symptom. There was also, it will be noted, a palsy of the left side of the tongue at the same period, still further confusing the diagnosis. There was no doubt as to the position of the lesion but it was difficult to conceive of a tumor which would attack at the same time the left sixth and tenth, the right seventh nerves and the pyramidal tracts on the right side of the pons. It is just this irregularity, however, which is characteristic of pontine tumors when of a gliomatous nature, and to which I especially wish to call attention here.

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[3] Ladame: *Arch. Gén. de Méd.*, 1865, Vol. II., p. 129.

[4] Bernhardt: Beitr. z. Sympt. d. Hirngeschwülste. 1881, p. 191.

[5] Nothnagel: Specielle Path. u Ther. Vol. IX., No. 2. p. 148.

Crossed hemiplegia occurred in 19 cases of the 45 studied. Paralysis of one or more of the cranial nerves on one side and hemiplegia on the opposite side is the usual picture. Almost any combination occurs with equal frequency. The facial nerve, however, was paralyzed in 12 of these 17 cases. More rarely the third, fifth, sixth or seventh alone was paralyzed.

A study of the statistics shows that hemiplegia occurred with paralysis of the contralateral third and seventh in three cases, the third and sixth in two cases, the third, sixth and seventh in one case, the third, fifth and seventh in one case, the third, seventh and eighth in one case, the sixth and seventh in three cases, the fifth and seventh in one case, the third, the fifth and the seventh alone each in one case, the sixth in two cases. Considering the special nerves outside of their association with alternate hemiplegia, it will be observed that the third nerve was paralyzed in 16 cases.

The pupils were dilated in 9 cases, were irregular in 4 cases, and were contracted on one side or both sides in 4 instances. The pupils did not react to light, or but little, in 2 cases and did not react in accommodation in 2 cases. Nystagmus was present in 7 cases and exophthalmos in one case. Conjugate deviation of the eyes was present in 5 cases. The importance of this symptom has been emphasized by Bernhardt who stated that if conjugate deviation to one or the other side persisted, added to palsy of the extremities, contralateral to the abducens palsy, it constituted a certain diagnostic sign of tumor of the pons.

A symptom of great interest, and one which perhaps has not been given the importance which it deserves, is paralysis of associated movements of the eyeballs. Paralysis of associated upward and downward movements was present in three cases (Diller[6] and Spiller), and paralysis of associated lateral movements was noted in 6 cases (Ewald,[7] Ormerod,[8] Ashworth,[9] Wernicke,[10] Jolly[11] and Gee[12]). This symptom is of great diagnostic importance.

Foville and Desnos[13] were the first to describe paralysis of associated movements in pontine tumor and Nothnagel noted the fact that paralysis of associated lateral movements was not rare. Attention should be directed here, to the studies of associated eye movements by Spiller, who believed that "persisting paralysis of associated lateral movement indicated a lesion of the posterior longitudinal bundle," and that "persistent paralysis of associated upward and downward movement indicates a lesion in the vicinity of the oculomotor nucleus." These are important facts in relation to the diagnosis of focal lesions of the pons. There was only a specific note of paralysis of the fourth nerve in three cases. The fifth nerve was found involved in 12 cases, which showed either anesthesia or anesthesia added to paralysis of the masseter muscles. The motor portion of the fifth nerve appears to be less frequently involved than the sensory portion. Loss of sensation of the face on the side opposite to the lesion occurs if the ascending roots of the fifth nerves are implicated, the motor symptoms not being present as has been mentioned by Bernhardt. The sixth nerve was paralyzed on one or both sides in 12 cases. The facial nerve was implicated on one or both sides in 34 cases.

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[6] Diller: *Am. Jour. Med. Sci.*, 1892, No. 104, p. 509.

[7] Ewald: *Berl. klin. Wochnschr.*, 1910, Vol. 39, p. 839.

[8] Ormerod: Tr. Path. Soc., London, 1890, Vol. XLI., p. 2.

[9] Ashworth: *Australasian Med. Gaz.*, 1896, No. 15, p. 295.

[10] Wernicke: *Arch. f. Psychiat.*, 1877, No. 7, p. 513.

[11] Jolly: *Arch. f. Psychiat.*, 1894, No. 26, p. 619.

[12] Gee: St. Barth. Hosp. Reports, 1881, Vol. XVII., p. 287.

[13] Foville and Desnos: In Bernhardt.

Sense of hearing was noted as being disturbed on one or both sides in 15 cases. Bernhardt found implications of the eighth nerve in 8 of 19 cases. It would seem therefore that this is not a rare symptom as is claimed by Nothnagel. Sense of taste was diminished in 3 cases and paresthesia was noted in one case. Nothnagel looked upon this also as a rare symptom and Bernhardt found the sense of taste disturbed in 3 cases. Sense of smell showed subjective diminution in 1 case.

Dysarthria is a usual symptom of tumor of the pons. (Nothnagel, Delbanco,[14] von Monakow[15].) Speech was altered in various ways in 29 cases. It was noted as being either thick, blurred, bulbar, nasal or was otherwise disturbed. This is an important symptom, Bernhardt having found it present in 13 of his 19 cases. The tongue was protruded to the right or left in 7 instances, and Nothnagel states that the tongue is usually pushed to the side of the paralysis. There was difficulty in swallowing in 22 cases, an unusually large number of cases considering the fact that Bernhardt found it present in only 4 of 19 cases.

A symptom to which attention has been called by Nothnagel, and which has been observed in a few cases, consists of pains in various parts of the body. Paresthesia was noted in 1 case, spreading from the left arm, right temple, to right side of head and down back of neck. In one case there was pain in the right shoulder and right intercostal nerves; in another case, pain in the left side; and still another, pain in the neck.

Paralysis of the limbs occurred frequently. There was weakness of the left arm in 9 cases; in the right arm, in 5 cases; and in both arms, in 1 case. Hemiplegia of one or the other side occurred in 12 cases. One or both legs were weak in 15 cases and there was general weakness in 15 cases. Rigidity of the arms and legs occurred in 2 cases. Ataxia occurred in 23 instances, affecting either the gait, station or movements of the arms and limbs. This would indicate that this symptom is more frequent than is generally believed for Nothnagel noted its presence in only a few cases.

Forced positions occurred in 8 instances, the head being drawn to the right in 5 cases and to the left in 3. In one case the head was bent forward in walking and in another the head was moved from side to side when the patient was sitting.

Nothing definite was noted in the study of the reflexes except that they were usually exaggerated. The knee jerks were increased on both sides in 20 cases, and were increased on one or the other side in 6 cases. The patellar reflexes were lost in 6 instances. Ankle clonus was present in 5 cases on both sides, and on one side in 4 cases. The Babinski phenomenon was present in 2 cases.

The bladder functions were disturbed only in 4 cases, consisting of incontinence or retention. Incontinence of the rectal functions was present in 4 cases. While Nothnagel has noted this symptom without disturbance of the sensorium, it can not be doubted that in most instances there is some accompanying disturbance of the consciousness.

Of the 45 cases studied, 14 were cases of glioma (Rendu,[16] Jolly, Wood,[17] Hoffman,[18] Jackson,[19] Williams,[20] Ashworth, Ross,[21] Vorkastner,[22] Henneberg,[23] Hudovernig,[24] Thomson,[25] Spiller, Rhein); 11

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[14] Delbanco: Beitr. z. Symp. u. diag. d. Geschwülste d. Pons Varolii Mang. Dis., 1891, Berlin.

[15] vonMonakow: Gehirn Path., 1904.

[16] Rendu: *La France Médicale*, 1889, Vol. I., p. 625.

[17] Wood: *Ophthalmic Record*, 1904, No. 13, p. 529.

[18] Hoffman: *Virchow Arch.*, 1896, No. 146, p. 509.

[19] Jackson: *Australian Med. Jour.*, 1893, No. 15, p. 245.

[20] Williams: *Brit. Medico-Chirurgical Jour.*, 1891, Vol. IX., p. 163.

[21] Ross: *Brit. Med. Jour.*, 1884, No. 1, p. 906.

[22] Vorkastner: *Neurol. Centralbl.*, 1905, Vol. XXIV., p. 133.

[23] Henneberg: *Neurol. Centralbl.*, 1904, Vol. XXIII., p. 24.

[24] Hudovernig: *Neurol. Centralbl.*, 1903, Vol. XXII., p. 177.

[25] Thomson: *Neurol. Centralbl.*, 1901, p. 619.

tubercles (Wernicke, MacGregor,[26] Kolisch,[27] Blocq and Guinon,[28] Hepworth,[29] Levi,[30] Ewald, Sano,[31] Finney,[32] Strozewski[33]); 8 sarcomas (Goodhart[34]; Hektoen[35]; Mann and Delepine[36]; Diller: Nonne,[37] fibrosarcoma; Hun,[38] gliosarcoma; Handford,[39] myxosarcoma; Weeks,[40] myxosarcoma); 3 gummas (Hall,[41] Mills,[42] Zahn[43]); 2 carcinoma (Ladame; Cufs,[44] adeno-carcinoma); 1 gumma or tubercle (Omerod); 1 cyst (Newton[45]); and 5 tumors were undiagnosed (Drake,[46] Sharkey,[47] Bristowe,[48] Touche,[49] Gee).

In conclusion, the symptoms characteristic of tumor of the pons are crossed hemiplegia, that is, palsy of one or more cranial nerves on one side, chiefly the fifth, sixth, seventh or eighth nerve, and hemiplegia on the opposite side.

The variability of the symptomatology on the other hand is also characteristic for obvious reasons when the tumor is an infiltrating glioma which is the most frequent type of pontine tumor.

The frequency of dysphagia and dysarthria should be emphasized.

Finally, paralysis of associated ocular movements are of extremely important diagnostic value as indicating the presence of lesion of the pons.

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[26] MacGregor: *Lancet*, London, 1889, No. 1, p. 1079.

[27] Kolisch: *Wien. klin. Wochnschr.*, 1893, No. 6, p. 252.

[28] Blocq and Guinon: *Arch. f. med. Exp. et Anat. Path.*, 1891, No. 1, p. 74.

[29] Hepworth: *Brit. Med. Jour.*, 1896, Vol. I., p. 16.

[30] Levi: Bull. Soc. Anat., Paris, 1897, Vol. LXXII., p. 514.

[31] Sano: *Jour. d. Neurol.*, 1899, Vol. IV., p. 288.

[32] Finney: *Dublin Jour. Med. Sc.*, 1889, Vol. LXXXVII., p. 380.

[33] Strozewski: *Neurol. Centralbl.*, 1900, p. 719.

[34] Goodhart: Trans. Path. Soc., London, 1886, Vol. XXXVII., p. 14.

[35] Hektoen: Med. Standard, 1893, No. 13-14, p. 70.

[36] Mann and Delépine: *Brain*, 1898, No. 21, p. 475.

[37] Nonne: *Neurol. Centralbl.*, 1907, p. 86.

[38] Hun: *Jour. Nerv. and Ment. Dis.*, 1887, No. 14, p. 624.

[39] Handford: *Brit. Med. Jour.*, 1898, No. 1, p. 1585.

[40] Weeks: Trans. Am. Surg. Assn., 1899, Vol. XVII., p. 218.

[41] Hall: *Lancet*, London, 1895, Vol. II., p. 263.

[42] Mills: *Jour. Nerv. and Ment.* Dis., 1881, No. 8, p. 470.

[43] Zahn: *Neurol. Centralbl.*, 1902, Vol. XXI., p. 364.

[44] Cufs: *Neurol. Centralbl.*, 1904, Vol. XXIII., p. 110.

[45] Newton: *New York Med. Jour.*, 1893, No.2, p. 766.

[46] Drake: *Phila Med. Jour.*, 1899, No. 4, p. 191.

[47] Sharkey: *Brain*, London, 1894, Vol. XVII., p. 238.

[48] Bristowe: *Brain*, London, 1891-2, Vol. XIV., p. 289.

[49] Touche: *Neurol. Centralbl.*, 1902, Vol. XXI., p. 42.

Unilateral Optic Atrophy and Contralateral Hemiplegia Consequent on Occlusion of the Cerebral Vessels

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UNILATERAL OPTIC ATROPHY AND CONTRALATERAL HEMIPLEGIA CONSEQUENT ON OCCLUSION OF THE CEREBRAL VESSELS

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Occlusion of one middle cerebral artery producing hemiplegia on the opposite side of the body is an everyday occurrence; furthermore. optic atrophy as a result of obliteration of the retinal vessels on the same side is well known, but the combination of unilateral optic atrophy and contralateral hemiplegia is seldom encountered and might be very confusing.

Cases of this kind have been described by Elschnig,[1] Gowers,[2] Hughlings Jackson,[3] Siegrist,[4] Williamson,[5] Batten and Guthrie,[6] Guthrie and Mayou,[7] Oppenheim,[8] von Monokow[9] and Starr.[10] With the exception of a brief reference in Starr's text-book, I have not been able to find any description of this symptom-complex in American literature; therefore it seemed important to record the following cases.

The first case was seen in the service of Dr. John K. Mitchell at the Orthopedic Hospital and Infirmary for Nervous Diseases, and the second at the University Hospital in the service of Dr. Charles K. Mills. To them I wish to express my thanks for permission to make this report.

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[1] Elschnig: Arch. f. Augenheilk., 1892, xxiv, 65, Case 2.

[2] Gowers: Lancet, London, 1875, ii, 794.

[3] Jackson, Hughlings: Ophth. Hosp. Rep., London, 1864. i, 360.

[4] Siegrist: 27 Versamml. Ophthalmologisches Gesellschaft, Heidelburg, 1898, p. 10.

[5] Williamson. R. T.: Brit. Med. Jour., 1898, i, 1515.

[6] Batten and Guthrie: Tr. Clin. Soc. Lond., 1903. xxxvi, 52.

[7] Guthrie and Mayou: Proc. Roy. Soc. Med., 1907-1908, i, 180.

[8] Oppenheim: Lehrbuch der Nervenkrankheiten, 1908. ii, 939.

[9] Von Monokow: Gehirnpathologie, Wien, 1897, p. 838.

[10] Starr: Diseases of the Nervous System, 1910, p. 500.

CASE 1.—*History.*—M. M., a man aged 35, was admitted to the Orthopedic Hospital and Infirmary for Nervous Diseases, in the service of Dr. J. K. Mitchell, Jan. 8, 1911. Syphilis and alcohol were denied. About six weeks previous to admission the patient had had an acute illness, said to have been influenza. During that time, while confined to bed, he suddenly developed a partial loss of consciousness, lasting a few minutes, with weakness of the right side of the body, including the face, arm and leg; there was also dimness of vision in the left eye and aphasia. Previous to this attack the vision had been normal.

Examination.—There was nearly complete motor aphasia, but no evidence of sensory aphasia, marked weakness of the right side of the face, cerebral in type, with spastic paralysis of the right arm and leg. The tendon reflexes were increased and there was ankle-clonus and Babinski sign. Vision of the right eye was normal, but the left eye was totally blind. Sensation was impaired for touch throughout the paralyzed side of the body. The other cranial nerves were normal. The heart was normal in size and regular; the sounds though fairly good were lacking in muscular tone. Dr. Langdon, who repeatedly made examinations of the eyes, reported that the right eye, except for slight grayness of the disk, was normal. This was not the grayness of true atrophy. In the left eye the arteries were cut down to mere threads; indeed, the inferior temporal branch appeared like a fibrous cord. This, with the macular changes, can readily be caused by occlusion of the central artery. The Wassermann reaction was negative.

Except for blindness and optic atrophy on the same side as the vascular lesion, this case differs in no way from the most commonplace embolic apoplexy with resulting hemiplegia. The character of the onset and course of the apoplectic attack itself, as well as the character of the paralysis, were not unusual; the patient, however, immediately after having recovered from the shock of the initial apoplexy complained of dimness of vision on one side, which rapidly progressed to total blindness. At first an explanation was difficult, but an ophthalmoscopic examination revealed optic atrophy, with the characteristic empty, thread-like appearance of the retinal vessels.

The cases of Batten and Guthrie,[6] of Jackson[3] and of Gowers,[2] which were studied at necropsy, have proved that an embolic mass can be carried through the internal carotid artery into the middle cerebral vessel, producing apoplexy and hemiplegia; at the same time a portion can be swept into the ophthalmic artery, as it branches from the internal carotid, or it may even be lodged in the central artery of the retina, and thus produce sudden blindness and atrophy.

In most instances blindness develops suddenly, though in one of the cases described by Batten and Guthrie optic atrophy was probably not completed for nearly three months after the initial apoplexy; a portion of the occluding mass in the internal carotid vessel may have been subsequently swept into the right ophthalmic artery. Singer[11] found, while producing embolic apoplexy experimentally on guinea-pigs, that occlusion of one retinal artery and subsequent optic atrophy almost invariably occurred. Undoubtedly there must be some peculiar arrangement of the central vessels in guinea-pigs, explaining the frequency of this occurrence.

It is not difficult to understand how optic atrophy and hemiplegia can be produced, if we remember the manner in which the terminal branches of the internal carotid vessels are distributed. It divides into the middle cerebral, which supplies the basal ganglia, internal capsule, etc.; close to the point where the middle cerebral is given off the ophthalmic is derived, which divides into two main groups, the orbital and ocular groups. The second group consists of the muscular, anterior ciliary, long and short ciliary and the central artery of the retina, the latter perhaps the smallest of all and possibly more easily occluded than the other branches.

Naturally, the position of the occlusion in the ophthalmic artery or in one of its branches has an important bearing on the resulting signs. Unilaterality of visual disturbances is in itself an important indication of a localized vascular lesion. In syphilis bilateral and symmetrical involvement would generally occur, or, at least, if one eye was affected, the other would show some evidence of disease even though relatively insignificant. Williamson has described unilateral retinal changes occurring with cerebral hemorrhage, embolism and thrombosis, in thirteen cases. In eleven the diagnosis was verified by post-mortem examination, and in two the diagnosis seemed correct clinically. In four cases of hemorrhage and one of embolism a large retinal hemorrhage occurred on the same side as the cerebral lesion, while the retina of the other eye was practically normal.

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[11] Singer: Ztschr. f. Heilk., 1897, xviii, 105.

In one case the retinal vessels were markedly dilated and tortuous on the side of the lesion.

Oppenheim,[8] in his text-book, speaks of an instance in which there was unilateral optic atrophy with contralateral hemiplegia, accompanied by considerable headache and tenderness on the side of the atrophy. He believed that it was caused by a tumor, but the necropsy revealed cerebral softening. Indeed, tumor might easily be confused, particularly when it is situated in or near the frontal lobe, as in the cases described by Gowers, Horsley and Kennedy.

For diagnostic purposes the appearance of optic atrophy when caused by vascular occlusion is more or less significant. Herbert Parsons[12] says:

> A large mass of evidence has already accumulated to show that of all parts of the ocular circulation the part in the immediate vicinity of the lamina cribrosa is the most liable to be affected by vascular disease in any form. This undoubtedly is true in regard to obstruction of the central artery whether it be due to embolus, endarteritis or endarteritis combined with thrombosis.

Parsons divides the appearance of the various stages of atrophy as follows: at first, the arteries are thread-like from more or less sudden cutting off of the blood-stream; later, there is some refilling from the establishment of a collateral circulation, and finally, the arteries are thread-like and sclerosed, associated with optic atrophy.

Unilateral optic atrophy may occur as the result of localized endarteritis or a combination of arteritis with thrombosis, without hemiplegia; but apoplexy and hemiplegia can develop subsequently, and then examination will show unilateral optic atrophy with contralateral hemiplegia; nevertheless an accurate history of the time of onset of blindness and a careful ophthalmoscopic examination should make the diagnosis possible. This condition is well illustrated by the following case:

CASE 2.—*History.*—P. R., a man aged 52, was admitted to the University Hospital, in the service of Dr. Mills, Nov. 15, 1911. Family history was negative. Patient denied syphilitic infection and alcohol. During 1909 and 1910 he had complained of dimness of vision on the left side. In February, 1910, he had a series of general convulsions. Three days later he suddenly lost consciousness. After regaining consciousness he was unable to speak and was paralyzed on the right side of the body.

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[12] Parsons, Herbert: The Pathology of the Eye, pp. 1262-1265.

Examination.—There was typical right hemiplegia with spasticity, increased tendon reflexes, ankle-clonus and Babinski sign and nearly complete motor aphasia. Sensation was not affected. On the left side there was complete blindness. Ophthalmoscopic examination showed optic atrophy with great diminution in the size of the retinal vessels, which were sclerotic. The retinal vessels of the right eye were moderately sclerotic, but vision was not much impaired.

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A CLINICOPATHOLOGIC STUDY OF CARBON MONOXID POISONING *

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AND

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PHILADELPHIA

REMARKS BY DR. MC CONNELL

Through the courtesy of Dr. Charles K. Mills, I am enabled to make this report of a fatal case of poisoning by carbon monoxid, the patient having died in his service at the Philadelphia General Hospital. As a recital of mere symptoms is almost valueless in these days of search for ultimate conditions, Dr. William G. Spiller kindly undertook the pathologic study and by his unusual findings established a sufficient reason for this presentation. I take this opportunity of expressing my appreciation of Dr. Spiller's kindness in associating his microscopic work with my clinical study.

Poisoning by carbon monoxid may be divided into three great classes, the division being determined by the mode of onset of the symptoms: acute poisoning, chronic poisoning and relapsing carbon monoxid poisoning.

Under acute poisoning are included those cases in which the manifestations appear very promptly after the exposure of the individual to the toxic agent, whether such exposure be accidental or intentional. The symptoms include nausea, an indefinite feeling of illness with fulness of the head, headache, vertigo, great muscular weakness, and, if the influence of the gas is continued, drowsiness, unconsciousness and loss of control of the sphincters. Muscular twitchings or even convul-

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* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

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sions sometimes occur. The skin and mucous membranes may be cyanotic and occasionally an erythematous rash resembling frost-bites may be seen over the malar prominences.

The sequelae of acute poisoning include pneumonia, cardiac palpitation, localized hyperemias, gastro-intestinal disturbances, transient glycosuria, cutaneous eruptions, localized edema and gangrene. In the nervous system, paralyses of central or peripheral type and choreiform movements are seen, persistent headache is complained of and mental changes take place, often only mild hallucinatory states but more commonly distinct confusional insanities.

Under the heading of chronic poisoning by carbon monoxid are placed those cases presenting headache, nausea, vertigo, general weakness and anemia with some slowness of intellectual activity and failure of memory. These are all symptoms of the acute type, but appear gradually in individuals who are chronically exposed to the toxic action of carbon monoxid.

The third class is designated as relapsing carbon monoxid poisoning, a term used to indicate the condition in those who, having apparently recovered from the initial effect of the poison, develop after a period of fair health a grave type of symptoms leading to death. Coplin, in 1905, explained the condition most uniquely, but without basis of fact. He said:

> In gas poisoning there takes place a combination of carbon monoxid with the hemoglobin of the hematoblasts present in the bone-marrow. The blood-making process is so crippled that when old injured cells are worn out and must be replaced there are no new cells prepared.

The occurrence of relapsing carbon monoxid poisoning was explained by Becker and Schwerin on the basis of a "deep-seated disturbance of the regeneration of all organs, especially of the vascular walls and the ganglion cells of the nervous system, as is evidenced by the secondary hemorrhages, idiocy, imbecility, etc., which are observed as sequelae."

Accounts of individuals suffering from relapsing carbon monoxid poisoning are not rare, and I could consume time and patience in reciting cases illustrative of the type of disease under discussion. One or two, however, that are strikingly similar will suffice for the purpose.

A man, aged 69, a porter, whose case was reported by Simon in 1868, was asphyxiated by carbon monoxid. His recovery was marked by persistent vertigo and headache. A month after his apparent return to health he developed a right hemiplegia quickly followed by death. Simon also reports the case of a woman, aged 46, who partially recovered from the acute effects of poisoning by carbon monoxid and subsequently developed mental disturbance, convulsions and rigid limbs. She was aphasic and died without any improvement in the nervous symptoms. Edsall calls attention to the fact that symptoms of much gravity often may be caused by a mild attack of poisoning, but these symptoms cannot be attributed to the immediate effects of the carbon monoxid, for Gruber, Sedgwick and Nichols and many others have fully demonstrated the non-persistence of the gas in the blood.

The following is the clinical report of a case which I observed for about six days:

CASE 1.—*History.*—The patient, C. S., was admitted to the alcoholic ward of the Philadelphia General Hospital in a stuporous condition, Nov. 28, 1911, supposedly suffering from alcoholism. There was no evidence of paralysis or other physical disability. As the patient did not improve she was transferred to the medical ward on December 1.

"She was listless, paying no attention to any one. She made no replies to questions and would not protrude her tongue. She was apparently conscious but had not the power to respond to requests. She would voluntarily open and close her eyelids and her eyes would follow the examiner as he walked about the bed. She seemed to understand some questions. Her mouth was very dry and she was given water from a spoon, but she made no attempt to swallow it."

Subsequent examination by Dr. Joseph Sailer was as follows: The patient was a poorly developed and ill-nourished woman of 62, without edema, jaundice, or enlarged thyroid. Over the cheeks and nose there was a diffuse erythema with a few pustules. The eyes showed equal pupils, slightly dilated with normal reactions. The tongue was dry and the teeth were poor. The pulse was rapid, regular and compressible. Save for a slight kyphosis the chest was normal in conformation and was everywhere resonant: the breath sounds were clear. The heart sounds were also clear but somewhat rapid, and there were no murmurs. The abdomen was retracted and the contents generally ptosed. There was marked spasticity of the upper extremities in flexion, and they could be passively extended only after effort. A similar condition of the lower extremities obtained but was less marked. The patellar tendon reflexes were absent. There was no ankle-clonus and the Babinski sign was not elicited.

Except for very slight mental improvement the condition of the patient remained unchanged and she was transferred to the neurologic wards on Dec. 13, 1911.

From her son it was learned that the patient had always been well; she had never had a sick day until two weeks before admission when she became mopey, dragged her feet, seemed tired and wanted to lie down. She talked, took nourishment, did not have pain and made no complaints. Her last two days at home were notable because of the loss of control over the bladder and bowels. About three weeks before admission she was overcome by coal-gas while sleeping. She was found unconscious and taken to a hospital where after ten hours she recovered consciousness, and in two days was discharged fully recovered and able to attend to her usual duties. This condition of good health lasted but a few days. Her family, past medical and social histories are immaterial. Her son said that she complained of the stiffness of the upper extremities, but attributed it to the artificial respiration movements employed at the hospital.

Examination.—Dec. 14, 1911, the patient was examined by Dr. Mills and myself. She was unconscious, lying on her left side, irresponsive to sensory stimuli, that is, she showed no change of countenance when pricked with a pin, although the plantar reflexes were very active. Her face had the appearance of general muscular contraction, giving to it a frowning or stern expression, a sort of squeezed expression. The corrugatores supercilli, the orbiculares palpebrarum and zygomatici muscles were apparently spastic. The jaw was tightly closed.

The malar-orbiculopalpebral and the fronto-orbiculopalpebral (McCarthy's) reflexes were very prompt. The left pupil was slightly larger than the right, which was irregular. Both reacted promptly to light.

The upper extremities were very spastic in the right-angle position at the elbows with the arms held close to the sides and the wrists and fingers semiflexed. All the deep reflexes, muscle- and tendon-jerks were plus. Those examined were the von Bechterew, the coracobrachial, the biceps, the triceps and the radiocarpal.

The abdominal reflexes were present. The patient was incontinent of urine and feces.

The right lower extremity was spastic in flexion at the knee and on the pelvis. The left lower extremity was spastic, less so than the right, and almost in extension. The deep reflexes were present and somewhat exaggerated, probably markedly so but restrained by spasticity. Delayed ankle-clonus, persistent and marked, continuing for some time, was present on both sides.

The plantar-metatarsal and metatarsophalangeal reflexes were marked. The small toes went down, but on careful selective minimal stimulation of the soles of the feet the great toes were extended more than their quiet position which was one of permanent Babinski.

Course.—The condition of the patient steadily became worse with the development of spasticity of the muscles of mastication, so that the stomach-tube feedings were supplanted by nasal feedings, which latter became unsuccessful because of regurgitation of the liquid nourishment and its entrance into the larynx.

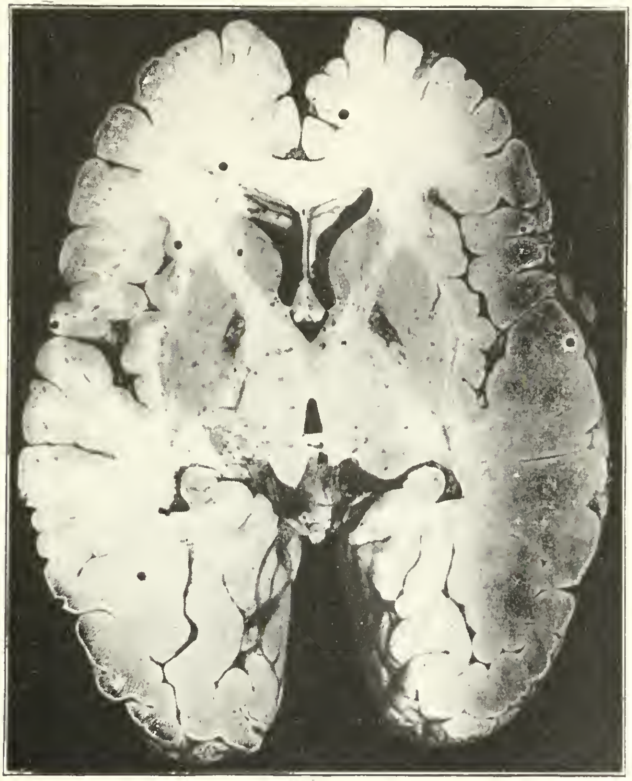

Fig. 1.—Symmetrical areas of softening in the inner segments of the lenticular nuclei.

The patient died on Dec. 17, 1911, nineteen days after admission to the hospital and forty days after the asphyxiation by carbon monoxid. During her residence in the hospital she had no fever except the terminal rise of temperature to 103 F. The pulse-rate was uniformly fast, averaging from 90 to 100 beats per minute. Respirations were between 20 and 25 per minute, until the final shallow breathing set in. The blood-pressure unfortunately was not measured, but at all times to the finger the pulse was soft and easily compressed.

Laboratory studies of the urine showed no albumin, no sugar and no abnormal microscopic findings. Examination of the blood gave 92 per cent. of hemoglobin, 5,890,000 red cells and 14.300 white cells. The Hoppe-Seyler test for carbon monoxid hemoglobin was negative, the resulting color being brown. This was not made, however, until six weeks after the intoxication.

A summary of the history of C. S. shows that about Nov. 7, 1911, she was asphyxiated by carbon monoxid and rendered unconscious, continuing so for ten hours, but recovering fully in two days. For a few days she was well and attended to her work; then she became "draggy," had some difficulty in locomotion and incontinence of urine and feces. On Nov. 28, 1911, three weeks after the poisoning, she was admitted to the hospital unconscious, universally spastic, with increased reflexes, without pulmonary, cardiac or trophic cutaneous symptoms. She died on Dec. 17, 1911, forty days after the inhalation of carbon monoxid. The account of the microscopic findings is given by Dr. Spiller.

The especial clinical interest in this case lies in the time which elapsed between the initial poisoning and the development of the grave symptoms, in the bilaterality of the motor symptoms, in the intense spasticity present and in the absence of distinct vasomotor or trophic symptoms.

As I have already stated, the weight of opinion seems to be in favor of the non-persistence of carbon monoxid in the blood and also the non-cumulative action of the gas. There must be, therefore, to explain the relapse, some physical or chemical process, originated by the poison and continuous in its action after recovery from the immediate effects of the acute intoxication. This I leave for Dr. Spiller to elaborate. In the paper by Dana on the functions of the corpora striata the statement is made that "in severe gas-poisoning there is a double softening of the lenticular nucleus due to thrombosis." This is the gross pathologic condition found in many of the reported cases, and fully explains the bilateral motor symptoms which conform to the type spoken of by Mills and Spiller in that the paralysis is less severe in lenticular disease than in capsular lesions.

Finally, many writers call attention to the distinct trophic cutaneous symptoms, but the only suggestion of a skin lesion in this case was the slight erythematous eruption over the cheeks and nose, somewhat like the appearance described by Peterson and Haines. Despite the fact that the comatose state lasted for days there was not a sign of decubitus or any gangrenous process.

REMARKS BY DR. SPILLER

The Betz cells in the case, reported from the clinical aspect by Dr. McConnell, are in a fair state of preservation, and there is little change in the paracentral lobules. The cells of the anterior horns of the spinal cord are well preserved, both in the cervical and lumbar regions. The vessels of the spinal pia and of the pia over the paracentral lobules and medulla oblongata are moderately thickened, and there is a very slight round-cell infiltration of the pia.

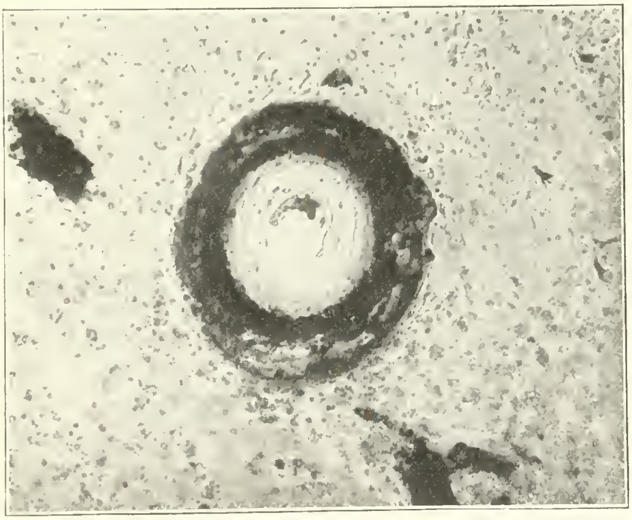

Fig. 2.—Calcified vessel in the area of softening in the lenticular nucleus.

The inner segment of each lenticular nucleus shows a well-defined area of softening (Fig. 1). These areas are almost exactly equal in size, and have almost exactly the same upper and lower limits. The vessels within these areas of softening show calcification of their walls (Fig. 2), so that they stain purple with hemalum, and black by the Weigert medullary sheath method. Numerous round or irregularly shaped masses, most of them having the structure of corpuscula arenacea, are found throughout the softened areas (Fig. 3).

The first thorough study of the encephalomalacia following carbon monoxid poisoning seems to have been made by Klebs; this honor is given to him by Hedrén.[1] Bilateral softening of the lenticular nucleus has been observed for many years as a result of carbon monoxid poisoning, and seems to be confined to the inner segments, but Oppenheim has written in the latest edition of his text-book that the method by which this softening is produced is not fully understood. The symmetrical softening of the lenticular nucleus always suggests carbon monoxid intoxication but it may occur from other causes, as Runeberg has shown (Hedrén). In this paper I report a case in which a history of gas-poisoning could not be obtained. The bilateral lesions were not quite so symmetrical as in the case in which a history of gas-poisoning was given.

Poelchen,[2] in 1888, was able to collect the reports of twelve cases of bilateral softening of the lenticular nucleus following carbon monoxid poisoning; at least, the inference is that the lesions were bilateral in all twelve cases. He also found similar changes in the inner segments of the lenticular nucleus and its vessels in a person dead from phosphorus-poisoning, so that phosphorus seems to have the same effect as carbon monoxid. The bilateral softening from carbon monoxid poisoning may be remarkably symmetrical, not only as regards the limitation to the inner segments of the lenticular nuclei, but also as regards extent of the lesions upward and downward. Poelchen's explanation of this is that the vessels of the inner segments of the lenticular nucleus are very narrow, even at their origin from the basal vessels, are without anastomosis, without vasa vasorum, and therefore dependent alone on the blood circulating within them. The arteries of the outer segment of the lenticular nucleus are comparatively large, and probably anastomose with the arteries of the island of Reil. If the carbon monoxid poisoning lasted long enough, other vessels would become affected.

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[1] Hedrén: Nord. med. Ark., 1902, ii, 4.

[2] Poelchen: Virchows Arch. f. path. Anat., cxii, 1888.

The most satisfactory explanation for the symmetrical softening has been given by Kolisko,[3] to whom we owe largely our knowledge of the circulation of this part of the brain. He states that in addition to the long arteries (arteries of cerebral softening supplying the head of the caudate nucleus, anterior limb of the internal capsule and anterior part of the external segment of the internal capsule) arising from the anterior cerebral artery a number of shorter arteries have their source in this artery which also convey the blood in a different direction from that in the parent stem. They are very small, about the thickness of a hair. Similar arteries arise from the carotid, the first part of the middle cerebral artery and the posterior communicating artery. These fine arteries do not anastomose. They supply the anterior part of both inner segments of the lenticular nucleus and the adjoining part of the internal capsule, including the knee. The extraordinary delicacy of these vessels with their almost perpendicular course at their origin is the cause of softening in carbon monoxid poisoning. In no other part of the brain is the circulation through

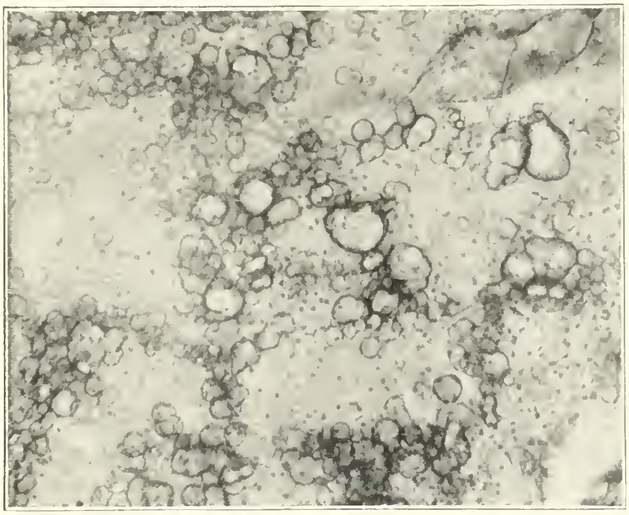

Fig. 3.—Calcareous deposits (corpuscula arenacea) in the areas of softening of the lenticular nuclei.

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[3] Kolisko: Wien. klin. Wchnschr., March 16, 1893, p. 191.

such short and narrow arteries. Kolisko has repeatedly had opportunity in necropsies on those who died a few days after carbon monoxid poisoning to find symmetrical softening in the distribution of these arteries. He thinks the great distention of these vessels, which occurs in the early stage of carbon monoxid poisoning and has been known since the time of Klebs, causes thrombosis and a disturbance of the circulation in these vessels.

Peterson and Haines[4] state that a large number of experiments on animals forced to breathe carbon monoxid, diluted with either oxygen or air, have resulted in showing that the blood-pressure is at first considerably increased as the result of the stimulation of the vasomotor center. A benumbing of this center is then shown by the decrease of the pressure and a distention of the blood-vessels.

From this it appears that Kolisko's arteries of cerebral thrombosis are not concerned in the softening of carbon monoxid poisoning, but as some of these fine vessels of which Kolisko speaks have the same course, the change in the direction of the blood-current affects them equally. Hemorrhage in the distribution of the arteries of cerebral thrombosis is exceedingly rare. Thrombosis, however, has rarely been found in cases of carbon monoxid poisoning. Poelchen, Sibelius[5] and other investigators have searched for it in vain, but it was found by Kolisko and by Hedrén.

Most extraordinary is the apparently complete or nearly complete recovery from the acute poisoning with recurrence of symptoms, possibly terminating fatally. The late symptoms may develop several weeks after apparent health. This recurrence of symptoms was observed in the case reported by Dr. McConnell, and similar examples may be found in the literature. I refer merely to one, as in the extreme spasticity it resembles the case we have studied.

Hedrén reports the case of a patient who, having been overcome by carbon monoxid poisoning, was brought into the hospital unconscious and after twelve days was dismissed apparently entirely recovered. He continued well three days and then began to have severe headache and pain in the extremities. Memory began to fail, general muscular rigidity and stupor developed, and death occurred about twenty-five days after the first return of symptoms. The rigidity was so great that the patient was brought from a lying to a sitting position with great difficulty, as he was "stiff as a board." Alteration of the vessels was found in the left lenticular nucleus with thrombosis of the small vessels of this part. Small areas of softening and small hemorrhages were found in the spinal cord.

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[4] Peterson and Haines: Text-Book of Legal Medicine and Toxicology, ii, 1904.

[5] Sibelius: Monatschr. f. Psychiat. u. Neurol., 1906, xiii, Erg.-Heft.

How are we to explain these delayed symptoms? There seems to be no support for the view that the occurrence of symptoms immediately or shortly after carbon monoxid poisoning depends on whether illuminating gas or coal-gas was the source of the intoxication. The carbon monoxid affects the nerve-cells as well as the nerve-fibers (Sibelius), and yet we can hardly attribute the delayed symptoms to changes in the nerve-cells alone. They were not conspicuous in the case reported in this paper. Delayed symptoms may depend in large measure on previous alteration of the vessels, as by age, and on the concentration of carbon monoxid in the gas breathed. Most cases of severe mental disturbance, in old persons, have been of the delayed type (Sibelius).

Carbon monoxid causes fatty degeneration of the intima and muscular coat of the vessels of the central nervous system. Sibelius attempts to give an explanation for the late symptoms, and attributes them especially to the alteration of vessels. The vascular changes progress after carbon monoxid has been removed from the blood, and carbon monoxid does not remain more than a few days in the blood. Wachholz, however, demonstrated the gas in the blood in a fatal case seven days after the poisoning. Sibelius thinks it is possible that from carbon monoxid poisoning several organs become affected, and that by imperfect elimination injurious metabolic substances are retained and affect the cerebral vessels. In some cases the vascular changes are not progressive, but their results are progressive from continued imperfect nutrition. Hemorrhages may occur and cause progressive symptoms, and yet hemorrhages have only occasionally been found. Aside from these possibilities we must assume, according to Sibelius, a progression of the vascular changes, although we do not understand the method. This I would explain, in part at least, by calcification. Sibelius makes no mention of calcification, and I do not know that anyone else has done so. The nearest approach to such an observation is by Poelchen. In a case in which he examined the vessels, drawn out of the lateral perforated substance at the base of the brain, which supply the inner part of the lenticular nucleus, he found fatty change in the intima and muscular coat, and in the larger vessels the muscular coat was calcified; all other vessels in the brain were normal. He does not mention calcification of the small vessels in the inner segments of the lenticular nucleus.

Calcification of the capillaries has been found in different parts of the brain under different conditions; also in the globus pallidus by Wollenberg (cited by Obersteiner). It was very pronounced in the inner segments of the lenticular nucleus in the case of gas-poisoning described in this paper, and was present in milder form in the same region in the case to be reported with similar symptoms but without a history of gas-poisoning, although the failure to obtain any history makes the etiology uncertain. In no reported case have I found mention of the deposit of calcium loose in the softened tissue or in the form of corpuscula arenacea, as in the case here described. This calcification of the small vessels of the inner part of the lenticular nuclei is probably a progressive process and as it increases impairs more and more the nutrition of the areas supplied by the affected vessels. It doubtless causes the progressive spasticity of the limbs, but it is difficult to explain the stupor from the limited lenticular lesions. It is worthy of note in this connection that in the cases of the family form of softening of the lenticular nuclei reported by Wilson the mental condition seems to have been peculiar.

I have sought an explanation for the formation of lime salts in the brain in Wells' "Chemical Pathology"[6] and find it stated there that in calcification the calcium salts come from the blood, where they are held in solution or in suspension by the proteids, either as the carbonate and phosphate themselves, or as calcium-ion-proteid compounds, or perhaps both. This suspension or solution is an unstable condition, possible only because of the extremely small proportion of calcium in the blood (about 1:10,000), and therefore capable of being overthrown by increased alkalinity of the blood, changes in the proteids or changes in the quantity or composition of the calcium salts.

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[6] Wells, H. Gideon: Chemical Pathology, W. B. Saunders Co., 1907.

Retrogressive changes in the tissues are a sine qua non. Hyaline degeneration, the chemical nature of which is not understood, is a very favorable condition, as also is necrosis when absorption is deficient.

In the areas that are to become calcified the circulation is very feeble, the blood-plasma seeping through the tissue as through any dead foreign substance of similar structure, without the presence of red corpuscles to permit of oxidative changes.

Calcification may be caused by utilization of the proteids of the fluids by the starved tissues so completely, because of its slow passage through them, that the calcium cannot be held longer in solution. This idea has little support but has been advanced. The possibility of the formation of calcium-binding substances within the degenerated area has always seemed more attractive, and has received the most attention by investigators.

Following is the report of a case which presented the same clinical appearance as did the case of gas-poisoning, but the patient was brought into the Philadelphia General Hospital without any history, and nothing could be learned of his previous condition.

Case 2.—E. B., aged 70, was admitted Dec. 22, 1911, and died Jan. 2, 1912. He was stuporous although he answered questions at times when spoken to, but his speech was incoherent. He muttered and sang in a low voice; he had no idea of place or time, was incontinent and said he heard and saw the Lord. Later he could not be aroused. The right pupil was very much dilated; the left was contracted and irregular. They did not respond to light. There was rigidity of all the limbs, especially at the elbows. The patient lay on his back with head and eyes turned to the right. Both forearms, especially the left, were strongly flexed on the arms. Extension of the forearms passively was difficult, and when the left was extended the patient screwed up his face as though in great pain. Pricking the face with a pin caused no change in expression. The lower limbs were slightly flexed at the knees but were more easily extended than the upper. The man moved the right arm and both legs at times quite freely, but did not move the left arm—at least no voluntary movement of this limb was noticed. The biceps and triceps reflexes were exaggerated. The patellar reflexes were not obtained, probably because of the spasticity. Plantar stimulation gave plantar flexion.

Symmetrical areas of softening were found in the inner segments of the lenticular nuclei. They were larger than in the case with a history of gas-poisoning, and did not have exactly the same superior and inferior limits. The small arteries in the areas of softening showed a moderate degree of calcification, but corpuscula arenacea or other calcium deposits were not found.

Dana[7] probably was the first American writer to call attention to the bilateral softening of the lenticular nucleus in gas-poisoning. The cases described in our report are especially interesting in connection with the extraordinary cases of progressive degeneration of the lenticular nucleus of the family form, described by S. A. K. Wilson.[8] It appears that when the lesions are confined to the lenticular nuclei intense spasticity is produced with apparent paralysis, but power of voluntary motion is really retained, although all movements are awkward and are of less power.

I am indebted to Dr. A. J. Smith for the photographs.

701 North Fortieth Street—2046 Chestnut Street.

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[7] Dana: Jour Nerv. and Ment. Dis., 1908, p. 65.

[8] Wilson, S. A. K.: Rev. Neurol., Feb. 29, 1912, p. 229; Brain, 1912, xxxiv.

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